



# MANUAL OF TROPICAL MEDICINE

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## PREFACE TO THE THIRD EDITION

THE second edition of this manual went out of print some years ago. The war however prevented us from preparing a revise at an earlier date and the same cause has introduced many difficulties in the preparation of this edition.

We have been compelled to rewrite large portions of the book and we have taken the opportunity to introduce certain subjects hitherto omitted and also greatly to increase the number of illustrations.

In order to keep down the size we have omitted the list of figures and the index of authors names. Notwithstanding this the manual has become somewhat unwieldy but we retain it in the form of one volume because our experience in the tropics makes us believe that this is the most convenient form for the tropical practitioner and student alike.

As regards nomenclature of parasites we have followed as in previous editions the rules of the International Committee. But regarding the names of the diseases we are using in most instances commonly known names.

Much of the work detailed therein is original and based upon life and experience in the tropics in which we have resided for periods of or exceeding two decades. We know how soon a work on tropical medicine becomes antiquated and we have ventured to go ahead as subjects which to-day are nebulous and attract little attention may become of general interest and importance in a few years.

We consider the mass of material which we have been compelled to handle in the preparation of this edition it is impossible to hope that we have not omitted reference to important facts

have not done injustice, however unintentionally, to some author who has not made errors of transliteration or otherwise, and for all these we ask the reader's indulgence, and beg him to remember that during the war it has been most difficult for us to obtain the time necessary for the preparation of this work.

We desire to record our grateful appreciation of the kind help received from Colonel Leiper Major Low and Professor Simpson.

We gratefully acknowledge the kindness of the following authors or their proxies, as war conditions have prevented our direct communication in giving us permission to copy illustrations: General Sir Havelock Charles Colonel Sir James Cantlie Colonel Balfour Colonel Wenyon, Colonel Stephens, Colonel Richard B. Strong, Major Broughton Alcock Captain O'Connor Dr Christopherson, Princess de Poix Mr Wellcome Professor Pinoy Professor Legroux Professor Hewlett Dr Sambon, Dr James Mr Hirst Miss Carter, Dr G. C. Shattock Dr Jackson, Dr Lutz and Dr Guilhaumon.

We more especially desire to thankfully acknowledge the generosity with which Dr J. J. Bell has placed so many of his valuable photo-micrographs at our disposal.

We have much pleasure in acknowledging our indebtedness to the *Tropical Diseases Bulletin*, which has been invaluable to us.

The index has been prepared by Miss James, to whom we tender our best thanks.

Finally, we wish to acknowledge the constant kindness and courtesy which we have received from our publishers Messrs Baillière, Tindall and Cox.

ALDO CASTELLANI  
ALBERT J. CHALMERS

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## CHAPTER I

# THE HISTORY OF TROPICAL MEDICINE

Primitive medicine—Accadia—China—Japan—America—Foundations of medicine—Indian—Egyptian—Jewish—Grecian—Alexandrian—Græco-Roman—Byzantine—Arabian—Medieval—Foundations of modern medicine—The discovery of the tropics—Early tropical medicine—Foundations of tropical medicine—References

tropical medicine—References

### Primitive Medicine.

PRIMITIVE peoples from the earliest times had some knowledge of medicine but they did not understand the phenomena of disease which they attributed to supernatural causes generally to evil or offended spirits. Hence in order to cure their ailments it was natural that they should seek to propitiate these spirits and accordingly we find that the medicine of primitive peoples was part of their religion and was administered by their witch doctors, fetichmen and priests.

This primitive condition is still met with in many parts of the

In Ceylon the superstitions are quite as elaborate as in West Africa if not more so. Pestilences are considered to be punishments on the people for

bad omen



perfection. It is beyond our space to enter into this interesting work of Chemin

condition among the North American Indians but appears to be advanced considerably among the Aztecs of Mexico and the Incas of Peru although very little is now known of the condition of knowledge among these peoples because the Spaniards destroyed all the records they could obtain. It appears that there were public hospitals in Mexico surgeons for the armies and a knowledge of circumcision venesection medicines and chemistry. The advent of the Spaniards while destroying our sources of history brought America once for all under European influence and the history of medicine therein forms part of the general advance of medical knowledge.

### Foundations of Medicine

Two races however appear to have advanced far beyond their elementary stage and to have laid the foundation upon which modern medicine has been built. These two races are the peoples of India and Egypt.

**Indian Medicine**—In India there are signs of the existence of peoples among whom at a very early period a better race from the north west forced its way. This race is often spoken of as the Aryan stock. Its earliest literature appears to have been in the

sons of the sun Surya who in their turn gave them to Indra. Indra taught Bharadwaja a learned sage who is said to be the author of the twelfth hymn of the tenth book of the Atharvaveda which belongs to the primitive age of the priest physician.

Bharadwaja taught Atreya who may perhaps be called the first physician of India as he taught medicine in Taxila somewhere about the sixth century B.C. Six of Atreya's pupils wrote compendia of his teachings of which only a single manuscript by Bhela (or Bheda) and a work by Agnivesa exist. This latter has however been edited by Charaka of Kashmir who left it unfinished when he died possibly in the second century A.D. This unfinished work was revised and completed by another Kashmir physician (Dridhabala) who used also the works of Vajbhata and Madhav. The book so compounded is the celebrated Charaka Samhita.

which after his period was forgotten till the eighteenth century, but he seems to have failed to recognize infection.

The works of Hippocrates who is justly considered to be the father of medicine are of a very high standard but it is probable that directly or indirectly he owed much to Indian and Egyptian influences.

**Alexandrian Medicine**—War produces great changes in the social life of nations and no exception is made for that portion which deals with disease. The wars of Alexander the Great led to the foundation of the city of Alexandria in the year 331 B.C. and this was followed by the transference of the headquarters of medical knowledge from Greece to Egypt where this knowledge was handed along the systematic lines laid down by Aristotle.

The result of this was that anatomy, pathological anatomy and medical jurisprudence progressed hand in hand with zoology and botany. Herodotus in 470 B.C. Agatharchides described *Druconculus*.

Under the Ptolemies medicine flourished but with the fall of Ptolemy came the end of the first and by far the greater period of Alexandrian medicine but its subsequent history is curious and interesting. Before the end of the great period Alexandrian medicine had found its way into Mesopotamia and thence into Persia which previously had been under the influence of Accadian medicine handed down by Babylonian and Assyria. Centuries when Alexandrian medicine had fallen to a very low level a new and stirring spirit by Syrians driven to Alexandria by Roman invasion of their country in the days of Heraculus. The result was that Syrian medicine took hold of the city and appeared in the Syrian language. Thus in the seventh century of the present era a priest called Aaron translated into Arabic thirty treatises by Abū Ibraj while later Sergius added two more treatises to this number. This is the heyday of Syrian medicine and the much lesser period of Alexandrian medicine had long ago given place to Greco-Roman medicine.

**Greco-Roman Medicine**—After Alexandrian medicine came Greco-Roman medicine largely derived directly from the Greek or Roman medicine until this influence came to be felt was very primitive. Among the physicians of this period may be mentioned Herodotus of Iadicea (50 B.C.) who was the first to describe elephantiasis *græcorum* or leprosy.

After him comes the great master of Roman medicine Aulus Cornelius Celsus (25 B.C. to A.D. 45). It is quite possible that Celsus was not a medical man but whether or not, he has left behind him in his eight books of medicine a most valuable treatise. To him belongs the credit of clearly distinguishing two types of typhus in malarial fever—viz. a simple and a much graver form. Half a century later this was put upon a scientific basis by the researches of Marcellus Celsus, and Dignus in the same city, Rome. He also recognized the double quartan fever, and gave a description of elephantiasis by which he meant leprosy.

After Celsus medicine flourished in both the Eastern and the Western Empires. Among the many writers of this period attention may be drawn to Aretæus of Cappadocia (A.D. 30-90) who describes dysentery and gives a long account of elephantiasis which he considered to be contagious by the inspired air.

Græco-Roman medicine reaches its zenith in Galen who was born

the degeneration of the mind and body with consequent relaxations of morals led to mysticism to the respect for the authority of magic and of the supernatural which was to pave the way to the bigotry dogmatism and mental inertia of the Middle Ages

There is therefore no surprise in finding that in one thousand years of Imperial rule Byzantium produced only four compilations—viz those by Orbasius of Sardianus by Ætius of Amida (a town in Mesopotamia) by Alexander of Tralles and by Paul of Ægin and some lesser works among which may be mentioned that by Actuarius on the urine

All these works are of interest but perhaps that by Paul of

cholera tenesmus and dysentery. His fourth book is particularly interesting beginning with a description of elephantiasis græcorum or leprosy. It contains an account of broad and round worms especially ascarides and dracunculus and further describes the bites of snakes dogs spiders scorpions centipedes lizards crocodiles and other animals. There is also a description of the stings

Arabian medicine and

religious persecution from Byzantium to Edessa in Mesopotamia where he began the study of medicine. Pursued even here by religious hate he fled into Persia where he established the *Gunde shâpâr Medical College* wherein were trained the original founders of Arabian medicine.

Under the Bagdad Caliphs many Greek medical works were translated into Arabic by Vesue and Johannitus while under the

phes and thus made not merely a great discovery but one which ought to be in time of lasting benefit to mankind. The full development of the human parasite was found out by Grassi who also showed that only Anopheles are capable of transmitting the parasite. Ross and Grassi's most important discoveries have been verified and extended by many people—e.g. Marchiafava, Celli, Bignami, Dionisi, Daniels, Stephens and Christophers etc—but the account of their work will be given later. Thus out of the fevers all classed as malaria there issued a type clearly defined to which the term malaria must be restricted.

In 1901 Forde and Dutton discovered a trypanosome called by Dutton *Trypanosoma gambiense* in a case of a peculiar irregular fever in the Gambia. In 1902 Dutton and Todd observed this organism in several other cases presenting the same type of fever which became known at the time as trypanosome fever, Gambia fever or Dutton's disease (Laveran and Mesnil). In 1902 and 1903 Castellani found a trypanosome in the cerebrospinal fluid of cases of sleeping sickness and first associated it with the aetiology. Further investigation by Bruce, Nabarro and numerous observers in various regions of Africa confirmed and greatly extended this work. In 1903 Sambon and Brumpt independently promulgated the hypothesis that the human trypanosome was carried by a tsetse fly; in all probability the *Glossina palpalis* and Bruce and Nabarro experimentally proved that the *Trypanosoma cristellani* is in reality introduced into human beings by the bite of *Glossina palpalis*. Bruce and others considered the transmission as purely mechanical but the researches of Kleine show that the parasite undergoes true development in the body of the tsetse fly. In 1903 Castellani stated that man in analogy with the lower animals might be infected by several species of trypanosomes. In 1909

(Westwood)

described in the lower animals by Dutton and Todd, Cazalbon, Lingard, Ed and Et, Sergeant, Shillong, Marten, Zemmann and others.

In the meanwhile Colonel Sir W. B. Leishman in the year 1900 discovered some peculiar bodies in the spleen of a soldier who had died of what was called 'dum dum fever' but did not publish an account of his discovery till 1903 in which year Donovan also found the same parasitic bodies in Madras. This parasite was first considered to be a proplasma by Laveran and Mesnil and called *Piroplasma donovani* but Ross created a new genus for it using the term *Leishmania*. Wright of Boston found similar bodies in Oriental sore which he called *Helcosoma tropicum*. The knowledge of these bodies and the diseases they cause has been considerably extended by Christophers and by Wartzinowsky and Bogroff while a great advance was made by Rogers who in 1904

Indian Medical Service in the bed bug. In 1904 Laveran and Cathoire discovered a *Leishmania* in films from the spleen of a child

islands. Gabbri considers the disease to be identical with Indian kala azar.

In 1903 peculiar parasitic bodies certainly protozoa were discovered in rabies by Negri of Pavia. Negri's important discovery has been confirmed by many authors and in the tropics by Cornwall.

In this section may be described the discovery of the causes of disease due to spirochaetes the nature and relationship of which are not yet clearly known. Obermeyer as far back as 1873 described the spirochaete of relapsing fever which was thought to be spread by the bed bug. In 1904 Nabarro, Ross and Milne in Uganda discovered a spirochaete in the blood of persons suffering from tick fever—the fever supposed to be due to the bite of in the same year Dutton bed a spirochaete causing ved by Breinl and King

horn to be distinct from the *Spiroschaudinna recurrentis* and in honour of the late Dr

covered a spirochæte in Weil's disease which they called *S. ictero hæmorrhagica*. In 1918 Noguchi cultivated from cases of yellow fever a spirochæte which he has named *Leptospira icteroides*. Other

same year Castellani demonstrated the presence of a spirochæte or treponema in yaws and named it *Spirochæta pertenue* (*Treponema pertenue*) and in 1906 described Bronchospirochætosus.

U

been worked out by Christophers. At the present time thanks to the researches of Koch, Theiler, Franca and others several

spreader of the disease

**Mycology**—Pathogenic mycology takes its origin in 1677 with

in 1844 that ringworm was due to a parasitic fungus and to the extension of that discovery by Malmsten in 1845 and to the great list of investigators of this particular feature down to the classical work of Sabouraud.

This work by Gruby produced world wide interest in parasitic mycology and for a time it made great progress but fell back into a second rate place when bacteriology came forward and is only

now being rescued from this position thanks to the labours of Manson Blanchard Bollinger Eyre Carter Vincent Nocard Pinoy and Brumpt

**Bacteriology**—From the most remote times the suspicion that the mysterious disease was due to minute organisms was sought in its origin by observers who were apparently popular in his time of the living nature of miasmata

Fracastoro's sixteenth century work is considered above while in 1641 Athanasius Kircher a friar stated that he had observed minute living organisms in the blood of a patient during an epidemic of plague Linnæus supported the theory that disease was due to minute forms of life by inserting papers on the subject in his *Amœnitates Academicæ* But the first to promulgate scientifically a bacterial theory was Agostino Bassi a country practitioner of the north of Italy in the early nineteenth century At that time a peculiar disease known as *silkworm disease* was prevalent in the country in which he lived and he discovered its cause by the means of the microscope He showed that the disease was due to the *microorganism* *Bacillus thuringiensis* *bassiana* From analogy Bassi believed and stated that human diseases were also due to microorganisms Bassi's work was not

to 1911 he introduced and improved methods for the separation and pure culture of bacteria and laid down the proofs required to

**Serums and Vaccines**—The discovery of the immune serums and their application to the treatment of disease marked a great step

Still more important are the labours of Roux Haffkine Wright Strong Lustig Galeotti and others in perfecting and applying vaccines to the prevention as well as to the treatment of disease

Those of greatest tropical importance are Haffkine's plague vaccine the same worker's cholera vaccine and Wright's monovalent typhoid vaccine and his pyogenic vaccines

A further advance was the introduction of Castellani's multiple vaccines for the prevention and treatment of disease. For long

prepared tetravaccines which include undulant or Malta fever and has prepared and advocated the use of penta and hexavalent vaccines

Entomology—It is obvious from the preceding sections that

ticks *Leishmania donovani* perhaps by bugs *Leishmania trisifatum* probably by fleas trypanosomes by tsetse flies and possibly by some kind of flies

But apart from these diseases of which we know the cause there are two infections the unknown agent of which is carried by mosquitoes. Thus Finlay in 1881 formulated definitely the hypothesis that yellow fever was spread by a mosquito which in 1900 was proved by Reed Carroll Agramonte and Lazear to be a fact the mosquito being *Stegomyia fasciata* now *Stegomyia calopus*

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A knowledge therefore of ticks biting flies and other insects is of the greatest importance to the doctor who is to practise in the



tropics We refer the reader interested in this subject to the classical work of Nuttall on insects as carriers of disease in the Johns Hopkins Hospital Reports 1899

The idea that the house fly and its allies are capable of spreading disease originates from the time of Mercurialis who in 1577 suggested that the virus of plague might be disseminated by this means In 1666 Sydenham remarked that the presence of numerous flies in the summer indicated that there would be much sickness in the autumn while in 1808 Crawford stated that he believed insects to be the carriers of infection In 1853 Moore referred to flies as the possible carriers of cholera typhoid tuberculosis anthrax and leprosy In 1869 Raubert performed the first experiments showing that anthrax could be disseminated by flies Tizzoni and Cattani made observations on the spread of cholera by the same means Grassi and later Stiles demonstrated the possibility of the carriage of parasitic worms and one of us the transference of the *Treponema pertenue* by the same means Gayon in 1903 indicated the possibility of the dissemination of fungi by flies

**Toxicology**—Micro organisms are not the only causes of disease to be found in the tropics for poisons from plants and animals are also of the greatest importance

It has been shown that the most primitive peoples have definite  
how early

of snake venom begun by Prince Lucien Bonaparte in 1843 has been extended by Fayrer Martin Lamb Calmette Noguchi and many others and leave a fuller description of this and the history of other poisons to a later chapter

**Climatology**—Tropical medicine does not confine itself to diseases caused only by parasites and poisons for there are such conditions as heat stroke which are entirely due to physical causes and also there is the important question of the influence of tropical climates on man which must be dealt with in a later chapter

**Dietetics**.—But little work has so far been done with regard to this important subject in the tropics though pioneer struggles have been undertaken most successfully by McCay in India and his example deserves to be followed

to tropical diseases are to be found in these early works on travel Thus as Singer has pointed out De Oviedo in 1526 gives a reference to a disease bubas which we now know to include *Frambæsia tropica* a form of Leishmaniasis and probably a form of Blastomycosis In 1558 Thevet described the jigger as a little worm called 'Tom' which entered into the feet and wrote descriptions of *Frambæsia tropica* under the term 'Pians' In 1598 G W wrote an

Espinlas possibly due to the bite or sting of some venomous

fever malarial fevers dysenteries smallpox climatic bubo in India malaria endemic yellow fever dengue smallpox filariasis diarrhoeas dysentery and yaws in West Africa as indicated by D. L. F. in 1726 and by Aubrey in 1729 but these and many others are briefly mentioned at the end of this chapter under the heading Special Works on Tropical Medicine and need not be further described here

It is not possible for us to trace out in detail the history of treatment but we may briefly mention a few points with regard to quinine arsenic antimony thymol and emetine

**Treatment—Quinine**—In the seventeenth century the epoch making discovery of the value of cinchona bark in the treatment of malarial fevers took place

In 1638 the Countess of Chinchon wife of the fourth Count Viceroy of Peru after nine years residence in that country was seized with tertian malarial fever Don Lopez de Canizaries the Corregidor of Loxa hearing of this sent her a parcel of the bark of a tree called by the Indians of Loxa quina quina The duplication of the name of the tree is said to indicate that it has medical properties

The value of this bark in the treatment of fever appears to have been only known locally but was understood by the Spaniards in

from fever on her estates near Madrid Hence the bark was often known as *Pulvis comitissæ* In 1670 Jesuit missionaries sent some of the bark to Rome whence it was distributed throughout Europe by Cardinal de Lugo Hence the names Jesuit's or Cardinal's bark

pellet her name  
trife now rose  
s bark or not  
h the dramatic  
to be known

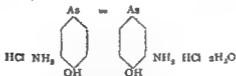
The tree has since that time been introduced into several parts of the world and grows well in India Ceylon and Java

Finally in 1820 Pelletier and Caventou prepared the alkaloid



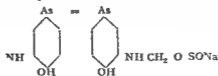
this combined treatment has apparently not given any better results than atoxyl alone. This combined treatment consisting of atoxyl and an inorganic salt of arsenic such as orpiment has been recommended by

diamino 4,4-dihydroxy arseno benzene—46—



On account of its phenolic and feebly basic properties Salvarsan fails to give neutral salts and the free base is very insoluble in water or in normal saline solution therefore an approximately neutral solution has to be prepared

arsenobenzene N-methylene sulphurate —



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in fact, cases of endemic malaria have already occurred, but we doubt whether there is any real danger of an epidemic. Bilharziosis has the opportunity to spread from Egypt to other countries. Are sufficiently strong measures being taken to combat the spread of these diseases and many others like them—*e g*, amœbic dysentery?

Another point which the war has brought into prominent notice is that so-called tropical diseases exist in abundance in Europe—

years of peace

It is not possible to close this history without acknowledging the debt which tropical medicine owes to the officers of the Royal Army Medical Corps, of the Royal Navy, the Indian Medical Service, the Colonial Service, and to their training schools, as well as to the officers of the Medical Services of the armies and navies of France, Italy, and the United States of America.

#### MODERN JOURNALS.

The very excellent *Tropical Diseases Bulletin* enables the tropical practi-

8 Annual Reports of the Principal Medical Officers of British Trench

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and

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53 Lister Institute Collected Papers (London)

54 Malaria e Malattie dei Paesi Caldi (Rome)

55 Malay Medical Journal

56 Malarologia (Naples)

57 Medicina Contemporanea (Lisbon)

58 Medizinischer Dienst in Nieder

Health of the Panama

(Washington)

62 Pacific Medical Journal

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65	Journal of Tropical
66	Proceedings of the Canal Zone Medical Association (Mount Hope Canal Zone)
67	Proceedings of the Society for Experimental Biology and Medicine (New York)
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The following journals have from time to time valuable papers on Tropical Medicine and Parasitology —

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6	Department (Washington)
7	Canadian Medical Association Journal (Toronto)
8	Collected Studies from the Research Laboratory Department of Health (City of New York)
9	Comptes Rendus de la Société de Biologie (Paris)
10	Deutsche Medizinische Wochenschrift (Berlin)
11	Johns Hopkins Bulletin

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24	Proceedings Series B and Transactions of the Royal Society of London
25	Quarterly Journal of Microscopical Science (London)
26	South African Medical Record, Cape Town

**JOURNALS WHICH HAVE CEASED PUBLICATION.**

Since the appearance of the second edition of this book the following journals have either ceased to be published as separate entities or have stopped publication altogether—

- 1 *American Journal of Tropical Diseases and Preventive Medicine* (This is now included in the *New Orleans Medical and Surgical Journal*) Three volumes were issued 1913-1916
- 2 *Journal of the London School of Tropical Medicine* Two volumes were issued 1911-1913
- 3 *Journal of Tropical Veterinary Science, Calcutta* Seven volumes appeared 1906-1912

volumes in existence which appeared from 1911 to 1915. Its work is carried on by the *Annals of Tropical Medicine and Parasitology*

**SPECIAL WORKS ON TROPICAL MEDICINE.**

(In Chronological Sequence)

DE CIVITATE ET REIPUBLICA... THE... DA...



- 63 Panama Canal Record (Balbão Heights Canal Zone)  
 64 Parasitology (Cambridge)  
 65 Philippine Journal of Science, Section B : Philippine Journal of Tropical  
 Medicine (Manila)  
 66 Proceedings of the Canal Zone Medical Association (Mount Hope, Canal  
 Zone)  
 67 Proceedings of the Society for Experimental Biology and Medicine (New  
 York)  
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The following journals have, from time to time, valuable papers on Tropical  
 Medicine and Parasitology —

- 1 Arbeiten aus dem Kaiserlichen Gesundheitsamte (Berlin)  
 2  
 3  
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 6  
 7 (Toronto) Department of Health  
 Laboratory  
 8 Comptes Rendus de la Société de Biologie (Paris)  
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**SPECIAL WORKS ON TROPICAL MEDICINE.**

*(In Chronological Sequence)*

DE OVIDO F. (1526). *Hystoria Natural de las Indias (Toledo)*. (1547)

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 SLOANE (1707-1725) A Voyage to the Islands of Madeira Barbadoes and

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 ALB The ... of the ...

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 MOSELEY B (1787) Treatise on Tropical Diseases (London) (Another  
 edition 1806)

HUNTER (1788)

BALFOUR I (1777)

THOMAS R. (1796)  
 (London)

JACKSON R (1791) The Fevers of Jamaica

WADE P (1793) Prevention and Treatment of the Disorders of Seamen  
 and Soldiers in Bengal (London)

HUNTER JOHN (1796) Observations on the Diseases of the Army in Jamaica  
 and on the Best Way of preserving the Health of Europeans in that  
 Climate (London)



- ... of the Origin Spread and Decline of the Epidemic  
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 MOORE, W J (1861?) *Manual of the Diseases of India* (London) Second  
 edition 1886  
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 LAVERAN A (1875) *Maladies et Epidémies des Armées* (Paris)  
 BACHOUÉ (1876) *Étude sur la Constitution Phys et Méd de Zanzibar*  
 (Paris)  
 ROY G C (1876) *Burdwan Fever* (London)  
 FOSSAGERIES J B (1877) *Traité d'Hygiène Navale* (Paris)  
 SULLIVAN JOHN (1877) *The Endemic Diseases of Tropical Climates*  
 (London).  
 BERENGER FERAUD (1878) *Traité Clinique des Maladies des Européens au*  
*Sénégal* (Paris)  
 HORTON J A B (1879) *Diseases of Tropical Climates* (London)  
 FAYRE JOSEPH (1881) *Tropical Diseases* (London)  
 BÉRENGER FERAUD (1881) *Traité Clinique des Maladies des Européens*  
*Pathologie Exotique* (Paris)  
 MACLEAN W C (1886)  
 BUROY (1886) *De la Fièvre dite bilieuse inflammation à la crujaan*  
 (Paris)  
 LE ROY DE MÉRICOURT ET CORRE *Du Traitement des Maladies Tropicales*  
*dans les climats tempérés* (Paris)  
 CORRE, A (1887) *Traité Clinique des Maladies des Pays Chauds* (Paris)  
 DUNCAN A (1888) *Prevention of Disease in Tropical and Subtropical*  
*Campaigns* (London)  
 KELSCH and MEYER (1889) *Traité des Maladies des Pays Chauds* (A  
 justly celebrated book)  
 DAVIDSON ANDREW (1893) *Hygiene and Diseases of Warm Climates*  
 FELKIN R W (1893) *Geographical Distribution of Tropical Diseases in*  
 Africa

- DESAINY CONSTANT (1893) Manuel de Médecine fifth edition (Hong Kong)  
(A work on the diseases of China with an extensive catalogue of drugs  
intended for missionaries)
- SCHUBERT B (1896) Die Krankheiten der Wärmen Lander (Jena) Second  
edition 1900 translated into English 1903 third edition 1903 fourth  
edition 1910

- TROPICANAUX (Paris)
- LE DANTEC A (1900) Précis de Pathologie Exotique (Paris) Third  
edition 1911
- REYNAUD G (1903) Hygiène des Établissements Coloniaux (Paris)
- AUDAIN (1904) Pathologie Intertropicale (Paris) (1910) Fièvres inter  
tropicales (Paris)

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## II Part II

- ... (1904 1912) Tropical Medicine and Hygiene 3 vols  
(London) Second edition 1914 1916
- JANSENE AND RIST (1909) Précis de Pathologie Exotique (Paris)
- GILLOU (1909) Manuel de Thérapeutique Clinique des Maladies Tropicales  
(Paris)
- SCHILLING C (1900) ...
- ... veral
- ... (1910)
- ... (London)
- ANDERSON (1918) The Epidemics of Mauritius (London)

## HISTORY OF TROPICAL MEDICINE

The most excellent book is GURRISON (1918) History of Medicine  
2nd edition Philadelphia and the important periodicals are Annals of  
Medical History commenced in 1917 and published in New York and the  
Archiv für Geschichte der Medizin started in Leipzig in 1908 and Janus  
as well as the Transactions of the Medical Historical Section of the Royal  
Society of Medicine

Neuberger (1910) History of Medicine vol 1 English translation by  
Playfair gives good accounts of Byzantine and Arabic medicine and Elliott  
(1914) Outlines of Greek and Roman Medicine London is most interesting

- ANDERSON (1908) Third Report Wellcome Tropical Research Laboratories 310 (1911) Fourth Report same Laboratories 248 (London)  
 These are most valuable observations upon Primitive Medicine in the Anglo Egyptian Sudan
- BERDOE (1913) The Origin and Growth of the Healing Art (London)  
 (Contains articles on the Medicine of Primitive Man of Egypt Judea Chaldea India Persia Mexico and Peru)
- CASTELLANI (1916) British Medical Journal 11 October 21 (London)  
 (Tartar Emetic and Protozoal Diseases)
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- February etc (Paris) (includes upon medicine and surgery)  
 Medicine)
- LABRY (1863) La Médecine chez les Chinois (Paris)
- DOROTHEA SINGER (1916) Transactions Royal Society of Medicine (London)  
 (Plague Tracts)
- GIMLETTE (1915)
- HEVISINGER (1839)
- JEE (1896) A S

- physicians)
- MORGAN (1918)
- ORIBASIS (1557)
- RHO Γ (1904)
- ROTH W E (18  
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- SINGER (1912) Annals of Tropical Medicine and Parasitology 6/3  
 (Liverpool) With D Singer (1917) Annals of Medical History 1  
 1 New York (Facastoro and Infection)
- SUGRUTA English translation (Calcutty) also a Latin translation about  
 1837 in Berlin

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## CHAPTER II

# TROPICAL RACES

The Tropics—Tropical Races—Primitive man—Classification—Laurasian division—Ethiopic division—Mongolic division—American division—References

### THE TROPICS

SUPAN suggested that climates should be classified as follows

1 *Tropical or warm climates* extending from the Equator to the mean annual isotherm of  $20^{\circ}\text{C}$  or  $68^{\circ}\text{F}$

2 *Temperate climates* extending from lands possessing a mean annual isotherm of  $20^{\circ}\text{C}$  or  $68^{\circ}\text{F}$  to those which have a temperature of  $50^{\circ}\text{F}$  for the warmest months of the year

3 *Cold climates* lying polewards of the isotherm of  $50^{\circ}\text{F}$  for the warmest months of the year

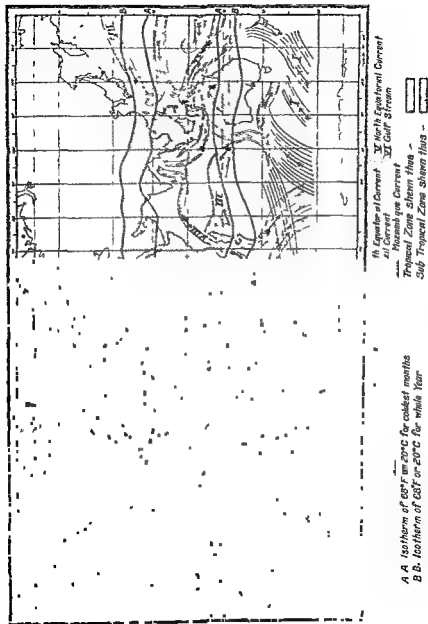
In Fig 1 we have depicted the tropical or warm climates as delineated by Supan's lines marked BB indicating the mean annual isotherm of  $20^{\circ}\text{C}$  or  $68^{\circ}\text{F}$  in north and south latitudes. Both lines are very wavy, the northern being situated about 35 degrees north latitude and the southern at rather less than 30 degrees south latitude. This region Supan has divided into two zones the *Tropical Zone* and the *Subtropical Zone* by lines indicated on Fig 1 as AA which represent isotherms of  $20^{\circ}\text{C}$  or  $68^{\circ}\text{F}$  for the coldest months of the year in both northern and southern latitudes.

These lines AA correspond more or less to  $33\frac{1}{2}$  degrees north and south latitude—i.e. more or less to the Tropics of Cancer and Capricorn.

1) 1 *The Tropical Zone*—Examining the map a little more in detail it will be noticed that starting in the extreme west and travelling eastwards the isotherm both north and south of the Equator is nearly the same as the latitude  $23\frac{1}{2}$  degrees north or south but as the coast of America is reached it dips a little to the south in the Northern Hemisphere and considerably to the north in the Southern Hemisphere. This variation is caused by cold currents in the sea running along the west coast of America, the more important of which (marked I on the map) is the cold current from the Antarctic.

Tracing the line farther eastward it will be noticed that it rises towards the north in the Northern Hemisphere and falls towards the south in the Southern Hemisphere. In the Northern Hemi-





sary to remember that though 25 per cent of the heat of the sun's rays which fall on the outer limit of our atmosphere are absorbed still these rays *do not really warm the air to any appreciable extent*. The real warmth of the air is obtained from the dark heat radiated from land and sea.

Land not only absorbs the radiant heat from the sun more quickly than water but also more readily gives off the dark heat to the air; therefore the presence of a large area of land upon which the sun's rays fall more or less vertically at noon all the year round will raise the temperature of the air, and will tend to extend the area of the warm climates. Hence the land may become extremely hot—incredibly high temperatures have been mentioned by authors—while water never becomes very warm. The reason of this is that water being a liquid by convection and by currents tends to keep at a more even temperature than land, the heat being distributed

Mozambique and the South Equatorial Currents may be noted and finally to the west and east of Australia the cold Antarctic

It will be observed that this zone includes Central America and

and the same isotherms for the mean temperature of the year

## TROPICAL RACES

It is now desirable to inquire very briefly into the races of man kind inhabiting these tropical or warm climates as defined above

with primitive man

## PRIMITIVE MAN

From geological, zoological and botanical considerations there can be little doubt that in early tertiary times there existed an *Indo-African continent* where at present the Indian Ocean lies. This continent embracing the Deccan, Madagascar and South Africa is more extensive than Scater's Lemuria and is now known as Gondwanaland.

This Indo African continent may for many reasons have been the site of the primitive home of the human race and indeed it was in Java that Dubois found those remarkable teeth, calvarium and femur which to day are recognized as belonging to *Pithecanthropus erectus* Dubois 1891 which geologically belongs more probably to the early Pleistocene rather than to the Tertiary Pliocene as was at one time considered possible. These remains belong either to a very early form of man or to an immediate precursor.

Once evolved there can be no doubt that the main factor in man's further evolution has been the development of the brain and this may have been stimulated by his remarkable migrations for driven by food requirements, geological or meteorological disturbances man migrated from his primal home and spread westwards into Africa where in the then fertile and well watered northern regions of the Sahara, Caucasian man probably evolved. He also migrated northwards into Asia, evolving there the common ancestor of Mongolic, Amerind man which eventually formed Mongolic man in Asia while the further migration into America gave

subside of the land to migrate westwards into Africa and eastwards into Oceania.

With regard to these early migrations it must not be forgotten that the climatic conditions were probably very different from those of to-day and as it was a warm interglacial period were distinctly favourable to these movements while the abundant land FAE o E me Asia to America and America to ated them Neither must well as subsequent migra tions were not single but multiple taking place in successive waves and spread over a long space of time.

When considering the different divisions of mankind in greater detail it will be noted that they spread from their original homes in various directions and at various times until the whole world was populated.

The first dispersal of man over the globe must have resembled the migrations of animals in that it must have been performed unconsciously under the influence of the factors just mentioned though it is possible that the food factor was the most potent because as Schigmann has pointed out the hunting man of to day requires a relatively large area in which to obtain his food and it is equally possible that primeval man soon found that a given district was unable to feed his rapidly increasing family or tribe. Under these circumstances the family or tribe in question would move into a more suitable region. When man became more evolved migrations would still take place under compulsion as described above but might also have taken place under the influence of attraction or expulsion by which one means that a powerful tribe might be attracted to an area held by a weaker tribe which latter would be compelled to submit to the conquerors or to migrate to some other area. If the weaker tribe remained with the stronger there would possibly be a race fusion as has so often taken place

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disease factor played in these migrations we do not know. What part epidemic parasitic diseases have played in extinction cannot be stated but that they must have played some part in the extinction of animals seems possible and it appears also possible that parasites transferred from animals to man at this period by

ruined the

mating

or broad

expanses of water and probably at these places settlements would be made from which reflux migrations into parts or finally occupied or passed through might arise due again to the influence of the

selection environment etc.

they had originally passed

and finding them more or less occupied by differently evolved peoples brought about fusion of the early divisions or subdivisions of mankind. And thus at an early period arose the first of these race fusions which are ethnologically so confusing to day. One factor in these early refluxes must be mentioned and this is the changed meteorological conditions brought about at the Glacial period or periods for during these man must have been driven towards the Equator while in the intervals he could wander polewards.

These early migrations and refluxes must have acted as potent

stimuli to the already rapidly evolving brain of man but this

specialized In the eastern part of this region arose the Accadians the Egyptians and the early Cretans from whom all the culture of Europe Asia and Africa evolved and separately in the west the Mexicans Peruvians Columbians and inhabitants of Yucatan whose advance was ended once for all by the Spanish conquests

and highly cultured Caucasian

The tropical regions of to day have therefore a most curious and most complex congeries of mankind First the indigenous inhabitants or natives of the land in question together with the

ethnology of man in the tropics is indeed complex but some elementary knowledge of the origin and relationships of the people among whom he is to work may be of use to the practitioner in the tropics and therefore we give the following brief classification leaving anyone interested in this subject an opportunity of further study by means of the works mentioned in the references at the end of the chapter

**Classification**—All classifications are more or less artificial and based upon the generally accepted knowledge of the day and are therefore ephemeral and the various classifications suggested by Bernier in 1684 Linnæus in 1735 1740 and 1758 Blumenbach in 1775 Virey in 1801 Des Moulins in 1825 26 Bory de Saint

Saint  
ca and  
finally

with the

- The Caucasian Division
- The Ethiopian or Negroid Division
- The Mongolic Division
- The Amerind Division

We will now briefly consider these divisions

**Caucasian Division.**

This division of man is thought to have evolved in Northern Africa at a time when the Sahara was a well watered and inhabitable region

**Characters** —The characters of Caucasian man are —*Height* average or above the average *colour* florid or pale *hair* long wavy soft and flaxen or long straight wiry and black in either case oval on transverse section *skull*

number 7,000,000  
inhabit the world  
increasing in number  
Europe 355,000  
15,000,000 Oceania 5,000,000

**Classification** —Ethnologically Caucasian man may be classified into the Xanthochroi and the Melanochroi while an early wave



stock is represented by the short dark broad nosed dolichocephalic peoples (recalling the noseless Daezu of the invading Aryans) but they have everywhere been modified by fusion with immigrant peoples giving rise to the Aryo Dravidian Scytho Dravidian and

principally

being

rather

while

complexion

of these people varies from medium brown to very dark and their noses from medium to broad. The Scytho Dravidian type of Western India is characterized by a fair complexion with little or no hair on the face with broad heads and moderately fine noses. Riseley considers that this type is a fusion between Dravidians and immigrant Scythians and Haddon suggests that it is a fusion with the Alpine race from the hills of South West Asia in prehistoric times. The Mongolo Dravidians are best represented by the inhabitants of Lower Bengal and Orissa. They are of medium stature and usually of dark complexion with abundant hair on the face with broad heads and broad or medium noses.

**INDONESIANS.**—The greatest divergence of opinion is found with regard to the inhabitants of Malaysia and Oceania. It would appear probable that the earliest inhabitants belonged to the undifferentiated negroid type of which the negritos gave rise to the Andamanese the Semangs of Malaysia the Aeta of the Philippines and the pygmies of New Guinea while the negroes formed the Tasmanians the Papuans and the majority of the Melanesians. In among these peoples came the brachycephalic Mongols called by Haddon the Proto Malays who were to be found first in the Peninsula and later driven south by the Caucasian migrations to be presently described in the islands.

Earlier migrations still perhaps of lowly developed Caucasian stocks may have given rise to the Pre Dravidian jungle tribes of India and Ceylon and perhaps to the Kakhiers of Northern Borneo and the Sakai of Malaysia and to one element in the Australian race.

An early migration fused with the Proto Malays forming the Proto Polynesians of Haddon who migrated into the Western Pacific and fusing with the early black peoples gave rise to the Melanesians while others passing through or round Melanesia went on to Tonga and Samoa and later to Tahiti and Raratonga of the Cook Islands spreading later to Hawaii and the Marquesas and still later to New Zealand. These migrations or voyages are supposed to have begun by a migration to Java as late as 65 B.C. and did not cease till A.D. 1350. In this way the mixed populations of many of the islands of the Pacific arose.



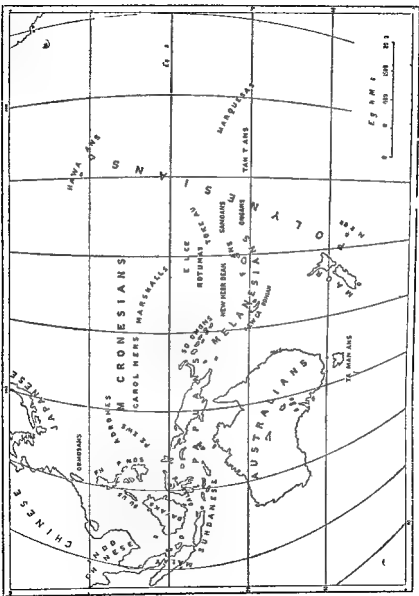


FIG. 3—THE RACES OF MALAYSIA AND OCEANIA  
(From Hitchcock's "The Races of Man")

## Ethiopic Division.

It is possible that this division took its origin in the Indo-African continent

Characters.—The characters of Ethiopic man are —*Height* either above the average (negro) or dwarfish (negrito) *colour* blackish or yellowish brown *hair* short frizzy, flat in section, or reddish brown and woolly *skull* dolicho

*Population*.—Ethiopic man is guessed to number some 175,000,000 of

origin, although others consider it safer to place them as intermediates between the Bantu peoples and the Bushmen. These Hottentots were stronger than

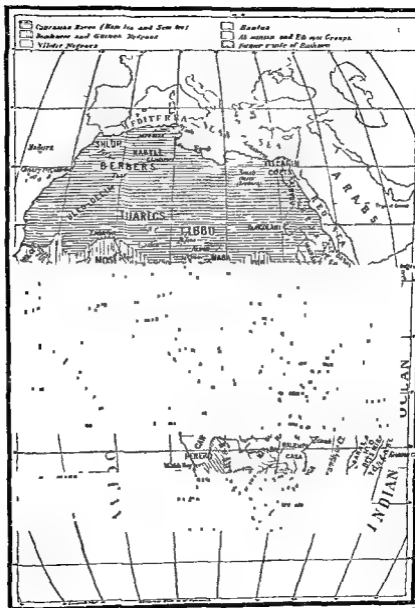


FIG 4—THE RACES OF AFRICA.  
(From Hutcheson's 'Living Races of Man')

The Ethiopians are divided into two principal sections—the Western or African and the Eastern or Oceanic section.

The stature of the Ethiopians of the Oceanic section is less than that of the Africans; the hair is more wavy, the nose is large and straight, the lips are not so thick, and are not everted as they are in the Africans. The Oceanics are more savage, but they show artistic taste and execute wood-carving.

The Western or African Section contains negritos and negroes. The negrito is dwarfish, with yellowish brown colour, reddish brown woolly hair, and brachycephalic skull, while the negro is tall of

the  
Ne  
and

## Mongolic Division.

Mongolic man probably evolved in the Tibetan Plateau of long ago

Population.	be 540 000 000
persons of w	55 000 000 in
Japan and	in Malaysia,
10 000 000 in	7 000 000 in
Turkestan an	200 elsewhere,

#### 4. *Urmiana*

The westward migrating Mongol Turks need not detain us except to state that they gave rise to the Turks proper the Samoyedes the Lapps the Magyars and Finns and the Bulgars probably only in part.

**Classification**—The Mongolic division (*vide* Figs 2 and 3) may be classified into—

1 *Mongols* who include the Mongols proper the Tunguses the Manchus the Koreans and Japanese

2 *Turkic Peoples* who are the Yakuts Kirghizes Turkomans

3 *Lapps Samoyedes Mordvins*

4 *Indo-Chinese* with the Tibetans Burmese Nagas Shans Siamese Annamese and Chinese

5 *Malayans* who are classed into the Malays proper the Javanese including the Sundanese Madurese and Javanese proper the Achinese Rejangs and Passimahs of Sumatra the Bugis Mangkassaras and Mmahasans of Celebes the Tagalas Bisayas etc of the Philippines the Dyaks of Borneo the Formosans and the Hovas of Madagascar

#### Amerind Division

The Amerind or American Indian division of mankind has a twofold origin from Europe and from Asia. From Europe dolicho-

way in Pleistocene times along  
the Orkneys the Shetlands  
land and Labrador These

peoples making their way across the continent met with later and more numerous arrivals the brachycephalic peoples of Mongol Amerind stock arriving from Asia by the land connections about the Behring Straits and the Aleutian Islands These two races fused and formed the Amerind division of man These Palæolithic races were apparently uninterrupted by any Caucasian Mongolic or Ethiopic migrations until the discovery of America by Columbus after which all three divisions made their migration thereunto Therefore all the culture of the Mayas Aztecs and Incas etc was an *inbred culture not dependable for its origin on outside sources* Hence the absence of the ordinary animals and plants of Asia Africa and Europe and the presence of peculiar animals and plants Hence also the presence of only stone and copper ages until the introduction of iron by the Caucasians and also the possible source of certain peculiar diseases such as yellow fever and perhaps *Francia tropica* and according to some authors syphilis which when introduced into Europe Asia and Africa produced such ravages

**Characters**—The characters of Amerind man are—*Height* above the average *colour* coppery or yellowish *hair* long coarse and black or section round *skull* mesocephalic *eyes* small round black sunken and

secluded valleys where alone traces of them can be found at the present time The Mexicans proper are the Otomi who are related to the Magahua and are to be found in the alleys of the Upper Mexico and in Guanajuato

herein

Passing into South America there is the great linguistic family of the  
Chibchas

out a permanent abode any great degree of civilization is impossible. The  
Chibchas also called Muyscans influenced the whole Panaman region as  
far north as the northern boundary of Costa Rica.

language

At the same time there existed near Truxillo the Chimu people speaking  
Mochica a language quite different from Quichuan. They reached to a  
degree of civilization but both they and the Quichuans were conquered by



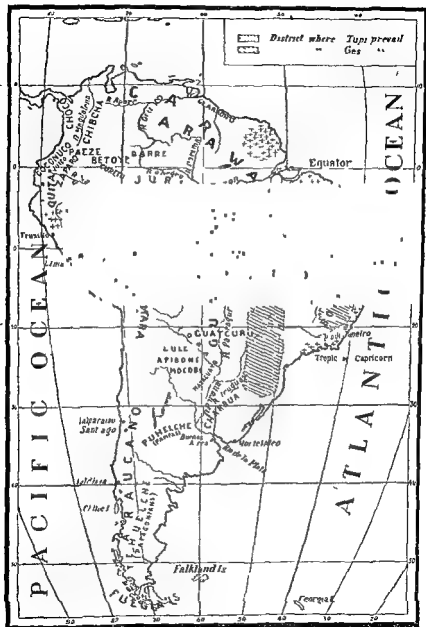


FIG 5—THE RACES OF SOUTH AMERICA  
(From Hutchinson's 'Living Races of Man')

The ... ..

No written language  
down from gener

th Amer ca and the  
peoples whom we now come to consider were all backward in their culture

The Arawak would appear to be the original inhabitants of the low lying lands to the east of the Cordilleras and it is possible that they originally spread to the north east the east and the south east from a primeval home on the eastern slopes of the Bolivian Cordilleras but their most important

family

Into the races mentioned above penetrated the great Caucasian migration headed by Columbus in the fifteenth century of our era which though

which are arising to-day



- HODGE (1907 ff) Handbook of American Indians North of Mexico  
Washington  
July to Decem  
resent (1906)
- London  
Institute Medical Research
- ondon " New Guinea  
Melanesians  
Anthropological Inst  
Journal Royal
- SELIGMANN C G (1906)  
(1910) Melanesians  
Anthropological Inst  
SELIGMANN C G and  
SPENCER AND GILLEN  
Australia  
London
- WOODRUFF (undated) Medical Ethnology London
- WORCESTER (1898) The Philippine Islands and their People

rapidly but when it is in the trade-wind belt from November or December to March or later the climate is dry

It is the northern migration of the Equatorial Belt which brings the heavy rains to the Uplands of Abyssinia and causes the rise of the Blue Nile and the Atbara which produce the rise of the Nile

The same features are to be noted in the movement southwards thus rain comes on the pampas of Brazil in the months from October to April while the dry season lasts for the rest of the year

Some few places—e.g. Wady Halfa—show only one maximum and one minimum temperature—i.e., the so called tropical type of temperature variation

**II Trade-wind Belts.**—The lands which lie just outside the polar boundaries of the equatorial or rain belt are situated some 20° to 35° north or south latitude and are among the driest in the world except in India where the south west monsoon brings a little rain into the dry regions of the Punjab and Sind

The worst places are the dry zones of California and other parts of North America the Sahara and Nubian Deserts parts of Arabia and Persia Argentina Eastern Patagonia South West Africa and the interior of Australia The only rain these regions are likely to get will come from the extension equatorially of the polar winds

We may therefore summarize the character of the trade-wind belts as very regular annual and diurnal ranges of temperature with a complete absence of rain or with slight showers at infrequent intervals The range of temperature in the desert is often very great thus during the day the temperature may be very high with dry winds carrying dust and sand and the nights with the clear sky free from cloud allowing active radiation may be cool if not cold, or even at times very cold

**III Monsoon Belts**—The word monsoon is believed to be derived from the Arabic word *Mansin* meaning a season

The monsoons (Fig 6) are classifiable into three groups—

- 1 North East and South West Monsoons
- 2 North West and South East Monsoons
- 3 West Monsoon

**1 North-East and South-West Monsoons**—These are typically met with in the Indian Ocean and its coasts

Dove's explanation of the *e* monsoons is generally accepted—viz that owing to heating of the great plains of Asia where the air ascends in the months of May June July and August the south east trade wind which is blowing south of the Equator is drawn northwards at the same time being deflected to the west thus forming the south west monsoon Conversely when the plains cool in November December January February and March there is a breeze from the north-east towards the Equator, which though called the north east monsoon is really a trade wind

The interval between the two monsoons is characterized by changeable winds which blow alternately in opposite directions—north east and south west

The south west monsoon is laden with moisture and on it Southern India largely depends for rain and a failure will mean a famine because a large portion of the population is agricultural for the natives depend for food upon

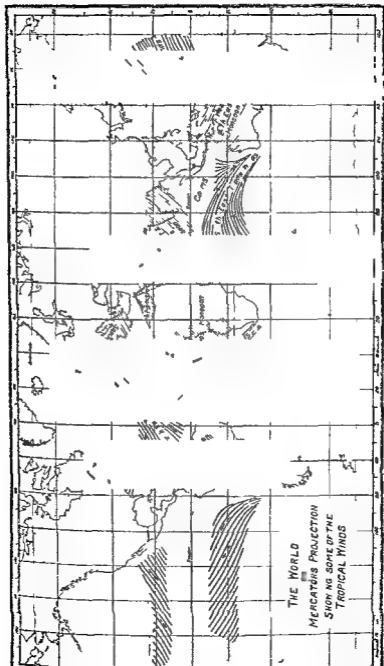


FIG 6—TROPICAL WINDS

their own cultivation and so densely are they packed that a failure to produce

minimum

It must be admitted that this simple classification like all classifications fails to explain everything and there are numerous exceptions showing combinations of equatorial trade wind and monsoon types.

**IV Mountain Climates**—We have already pointed out the effect of altitude in lowering the temperature and indeed Herschel many years ago showed that for every 300 feet of increase in altitude there was a decrease of  $1^{\circ} F$  in temperature and for every 180 metres a decrease of  $1^{\circ} C$ . These statements are only partially correct for the temperature at a given altitude depends upon the expansion and humidity of the air the clearness of the atmosphere together with the quantity of earth at the given locality and the nature of the wind blowing at the time of the observations. Perhaps

This will in part be counteracted by the heat produced by the condensation of aqueous vapour even at high altitudes and upon this condensation and cloud formation will depend how much of the heat radiating from the earth into the air is retained. The lessened amount of earth in high altitudes is also a factor as there is less earth to retain heat and hence less heat is given off into the air. It is obvious that movements of the air—the winds—must have a great effect depending upon their origin from warm or cold sources. Hence though in general the rule mentioned above as to a relationship between temperature and altitude holds good it is only approximate as the connection between the two is complex not simple.

In the tropics therefore the low country possesses the true tropical climate while the climatological conditions of the hills are quite different. Indeed as Ward has said the climates of many tropical plateaus and mountains have the reputation of having a perpetual spring but though that may be so at certain latitudes if considerable elevations are reached a perpetual winter exists with snow all the year round even on the Equator.

The great blessing of these elevations in the tropics is that they provide cool hill stations to which the resident of the plains may resort during the hottest months and in which sanatoria may be built

This brief sketch of tropical climates must suffice for our present purpose and we now turn to consider the various climatic factors and their effects upon man but the reader interested in this subject may find it useful to consult Chapter II pp 28 to 58 in our second edition in which more details will be found

## TEMPERATURE AND HUMIDITY

" "

" "

" "

"

rays but aqueous vapour is almost athermanous for infra red rays though largely diathermanous for other rays

In passing through the atmosphere about 25 per cent of the heat which has entered it is absorbed before it reaches sea level

The position of the sun is however of the utmost importance with regard to the quantity of heat reaching the earth If the sun is quite vertical probably only 20 per cent is lost whereas if

This atmospheric heat is one of the most important factors in determining the nature of a climate and shows daily and annual

It ought to be a wet season at these times and in their intervals a dry hot season

It is impossible however to consider the effects of temperature upon man without at the same time taking into account the humidity of the atmosphere

Man can bear very high temperatures easily provided the air is dry but not if there is much moisture or humidity in it The humidity of the atmosphere is in fact of the utmost importance in the study of climatic effects upon man This humidity is due to aqueous vapour caused by the constant evaporation which takes place from the surface of all collections of water

The humidity of the atmosphere presents three problems atmometry or the measurement of the quantity of water being taken into the air hygrometry or the determination of the quantity of aqueous vapour present in the air at any given time and hyetometry or the quantity of water being condensed from the atmosphere



Evaporation takes place most quickly in hot dry air and causes a considerable amount of heat to be rendered latent. This fact can be made use of in hot climates where it is a common practice to wrap flannel or felt round a bottle of water and after damping the flannel to hang the bottle in the breeze so that the contents may become cooled by the evaporation of the water from the flannel. The same principle applies to the porous stone or earthenware basins which are commonly used in Ceylon, Uganda, Egypt and other parts of the tropics to filter and cool water. Hence also the value of collections of water in keeping the temperature of places equable.

The amount of aqueous vapour in the atmosphere is generally spoken of as the humidity. Two kinds of humidity are recognized, absolute humidity which is the weight of aqueous vapour actually present in a definite volume of air at a given temperature and relative humidity which is the ratio of the weight of water actually present in a known volume of air to the weight of water which is required to saturate the same volume of air at the given tempera-

hotter but drier place. A fairly high relative humidity can however be borne if there is a breeze without which a much lower humidity is most unbearable. In fact Giles points out that Abusher in the Persian Gulf in August with a mean maximum temperature of  $96.5^{\circ}\text{F}$  ( $35.7^{\circ}\text{C}$ ) and a relative humidity of 65 per cent with no rainfall during the month and little or no breeze constitutes one of the most unbearable climates in the world though neither the temperature nor the humidity by themselves are high.

L. J. 41 14

hot in the tropics

### Effects

We must now consider briefly the effects of high air temperatures with and without high atmospheric humidity upon man and we will commence with a consideration of its effects upon the bodily temperature.

**Normal Temperature**—The normal temperature of man is the resultant between the heat produced by the oxidation of foodstuffs in the muscles glands and other organs and tissues of the body and the heat lost by warming the urine faeces and expired air by evaporation from the lungs and skin and by radiation and conduction from the skin. In other words the temperature of the body is controlled both by chemical and by physical heat regulation.

Factors affecting the loss of heat from the body are—

1. Temperature of the skin

- 1 Temperature and conductivity of the skin
- 2 Temperature and radiation from the skin
- 3 Temperature of the air
- 4 Rate of motion of the air

Clothing may however affect the loss of heat from the skin for as a rule

Air Temperature	Grammes of O <sub>2</sub> per Hour	Grammes of CO lost per Hour
2° C	37	29.8
10 to 15° C	28	25.1
15° to 20° C	19	44.1
20° to 25° C	23	25.0
25° to 30° C	43	5.3
30° to 35° C	84	23.7
35° to 40° C	112	21.2

This evaporation is very markedly diminished by humidity, as is shown in the following table —

Temperature in Centigrade	5 to 10 per Cent Humidity	81 to 89 per Cent Humidity
15.0°	16.3	9.0
20.4°	24.1	15.3
25.3°	75.5	23.9
28.4°	105.0	—

It is seen that

and the heat lost must vary considerably being the resultant between the heat gained and the heat lost.

In the

The methods of investigating the temperature of the body are by placing the thermometer—

- 1 In the axilla
- 2 In the mouth
- 3 In the rectum

to which might be added placenta, the thermometer in the urine but this is not done.

discussion but the methods are those who

time in India as —

Ten minutes in the well closed and dry axilla,  
Light minutes in the mouth  
Three to four minutes in the rectum

The mean daily temperature of man for the hours 2 a m to 12 midnight in the Temperate Zone is as follows —

98.45° F (36.90° C) in the axilla  
98.36° F (36.87° C) in the mouth  
98.96° F (37.20° C) in the rectum

The mean daily temperature of man in the Tropics is as follows —

98  
101  
101  
101  
101

At

Mean Morning Temperature	Mean Afternoon Temperature	Mean Twenty four Hours Temperature	Maximum Daily Range
97.763° F	98.341° F	98.084° F	1.41° F

But at present there are insufficient data upon which to base a definite conclusion as to the mean temperature of human beings for the whole twenty four hours

The average temperature of man in the Tropics is 97° F and 97° F (36.28° C)

37° C) the

**Effect on Temperature.**—Having now defined what we mean by a normal temperature in man it is necessary to inquire into the effects of high atmospheric temperatures and varying humidity upon man

The experiments of Linnæus (1734) Ellis (1754) Blagden and Fordyce (1775) established the fact that a normal man suitably clothed can regulate his

temperatures are 45° and 55°

sweating and a general feeling of exhaustion and discomfort

It was observations like these that caused Tyler to attempt to correlate personal sensations with meteorological data and to formulate his "hyther" degrees by which he meant the degree of discomfort caused by high air temperatures associated with high relative humidity

Haldane's experiments have often been repeated by ourselves on normal people by placing them alongside the condensers in the engine room of steamers in the Red Sea during very hot months. Near the condensers one can usually find a corner where there is no obvious movement of the air which is nearly saturated with aqueous vapour and where there is a high atmospheric temperature. Placed in such a corner and clad only in loose pyjamas the skin of the body flushes and burns; the perspiration rolls downwards in streams and the temperature rises.

This is the case in the engine rooms of steamships upon a return to cooler air.

If such a climate existed no human being could live therein.

discussed later.

From the time of Davy in 1839 observations have been made

of these results. The truth is that the earlier observers omitted

to take into consideration the atmospheric humidity and thus do not vitiate their conclusions

Our own observations agree with those who have failed to show any change in the bodily temperature in passages to and from the tropics and in residence therein provided that the individuals observed were normal

Further we have failed to observe any difference in the temperature between well nourished healthy natives and Europeans and with due allowance for individual and seasonal differences and the effects of exercise and clothing

In previous editions we have consumed much space with discussions as to the pulse rate and so as not to weary the reader we

tropical natives brought straight to temperate climates which tends to produce attacks of malaria liver troubles, and inflammation of the bronchi and lungs. He especially lays stress on temporary albuminuria yielding to treatment in some one to three weeks

**Effect on Respiration**—As we have no personal experiments to record with regard to respiration we give the work of other observers

Number of Persons 12	Temperature 65° F, Hygrometer 25° F	Temperature 78° F Hygrometer 4° F	Temperature 83° F Hygrometer 4° F	Temperature 65° F Hygrometer 15° F
Capacity of the chest as shown by spirometer	256.083	280.75	287.41	260.23
Gain or loss	—	+24.83	+0.5821	-20.333
Percentage gain	—	12.4	—	—

The gain of volume of Respiration is not due to any actual quantity excited

the lungs  
irritations  
per cent

reported  
after the

tropics Jousset however, states that this is only temporary, and disappears after acclimatization

Rattray maintained that there was a diminution of capacity when a native of the tropics went to the Temperate Zone and gave the following table —

Condition	Race	Temperature 79° F	Temperature 78° F	Temperature 32° F
B C aged twenty one, height 5 feet 5½ inches	Native of Sierra Leone	210	207	185
J C aged twenty height 5 feet 4 inches	Native of Sierra Leone	174	166	150
J W aged thirty one, height 5 feet 4 inches	Half c	174	162	—

He found that the frequency of respiration was slower in the tropics as is shown in the following table —

Climate	Mean Shade Temperature	Highest Number of Respirations	Lowest Number of Respirations	Average Number of Respirations
England	Summer 62° F	18	13.5	15.68
	Winter 47.25° F	17.5	15	16.50
Equatorial doldrum	Out ward 78.74° F	14.5	11	12.74
	Horiz ontal 76.60° F	14	12	13.74

Jousset, Plehn and others controvert Rattray's statement that the respirations are slower, believing on the contrary that they are augmented before acclimatization

Time	Number of Observa- tions	Tropics			Temperate Zone	
		Lowest	Highest	Range	Average	Average
9 a m	53	66	112	46	86.4	91.7
3 p m	53	68	108	42	88.8	88.1
9 p m	49	73	110	37	87.3	90.5
Averages	—	—	—	—	87.5	90.1

He gives the following conclusions —

1 The average pulse is lower by 25 beats in the Tropics than in the Temperate Zone

returns to normal

There is a total loss of tone in the arteries and veins

active Filipinos and Americans is a few beats above the usual standard of 72 per minute

to confirm this and find it to be the same as in Europeans

**Effect on the Blood**—Mitchell as the result of his work in the Persian Gulf considers that damp heat of itself frequently produces anaemia but gives no details of the blood examinations. In 1916 W. M. Strong noting that persons who had lived for long in the Tropics

amount of each spectral tint which is reflected back to the eye. Further he considers that when more pigment is deposited the skin becomes yellow brown.

The Arneth count (see p. 1898) in healthy native children has been investigated by Brenn and Priestley taking the figure for normal Europeans as 40. Native children varied from 71.6 to 83.86 while adult natives stood at 74.04. They are of the opinion that the alteration of the blood picture is the outcome of climatic influences and not as suggested by Scott Macfie as the outcome of abortive inoculation with malarial parasites though they think that the higher figure—viz. 83.86—may be accounted for by infection.

Sweet agrees that high Arneth index is very regularly present in healthy children over the whole coastal area of Eastern Australia. He maintains that no factor apart from disease is found definitely to influence this index but



As it descends in the region of subtropical calms it passes polewards or to the Equator. Within the arc of 30 degrees there are winds called the 'trades,' which from 30 degrees towards the Equator, and which on reference to Fig 6, can be noted as north-east trades and south-east trades and it will also be observed that the only portion of the ocean free from them is the North Indian Ocean, where the south-west monsoons occur. The north-east monsoon is really a trade wind.

Further, it will be noted that there are calms along the surface in an absence of prevailing winds.



FIG 7—DIAGRAM OF THE CIRCULATION OF THE ATMOSPHERE (After Ferrell)

... of Capricorn. Again at ... and these are often ... The trade-winds are only to be seen typically in oceanic regions, where the temperature is equable, and there are no local conditions to cause them to deviate from their course. But they, together

extend from 7 degrees to 29 degrees north in the Atlantic, and the south-east to 20 degrees south. During the summer they advance a few degrees north, and in the winter recede to the south. In ... of the doldrums is only 1 to 2 degrees north of

the Equator while in summer it is about 9 or 10 degrees north latitude and as will be explained later the tropical rains of certain regions depend upon this movement. Their easterly direction is due to the rotation of the world.

Periodical winds

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is very enervating

**The Solano**—The solano is a south easterly wind blowing from the Sahara into Spain.

The Pampero

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Andes

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Europeans

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**Pamperox**—These are the south westerly winds of Brazil. Europeans have altered the significance of the name to squally cyclonic winds in the same vicinity.

**Ghibli**—The ghibli is a violent south or south east wind blowing into Tripoli from the desert.

time that the wind came from the Sahara but all evidence is against this and the present idea is that it is a local wind which is produced by a high

on to the Canterbury Plains

ould have been most violent on the south coast whereas it is actually at its greatest violence on the north coast especially at Palermo

— — — — —

### Electrical Conditions

Silent electrical discharges are frequent in the tropics and the acidity which they produce is supposed to be the cause of devitrification which is so trying to persons working with all except the best microscopical and other lenses

field as far as we know for we have been unable to find any definite scientific observations which can confirm or refute the popular belief Hahn considers that there is no indication that atmospheric electricity plays a notable part in climatology

### Sun s Rays.

percentages —

	Per Cent
Ultra violet	39
Violet	42
Blue	48
Green sh blue	54
Yellow	63
Red	70
Infra red	76

Freer Gibbs and Bacon have shown that though the tropical light of Manila contains few if any more ultra violet rays than the Temperate Zone still the chemical rays have more effect there than in a temperate climate and these effects vary on different days. Freer distinguishes actinic and non actinic days on both of which the sky may be equally clear. The cause of the difference between an actinic and non actinic day is not understood. Gibbs does not believe that the normal intensities of the light in the tropics is different from that of any other region the

excluded the  
 suspects that  
 to the long  
 which we will

now consider

The chemical rays appear to have first a stimulative and beneficial influence and secondly a harmful influence

**Stimulative Influence**—Ginsen by experiments upon tadpoles earthworms beetles flies etc came to the conclusion that the action of the chemical or blue violet rays was very considerable as compared with light (yellow) or heat (red) rays and that though their action was probably very complex still it could be best considered as an excitation of the nervous system

This excitation was so powerful as to produce reflex actions in tadpoles and movements in other animals while in man he considers them to be the cause of the feeling of *bien etre* experienced on a bright sunny day which he compares with the depression felt on a dark cloudy day

**Bactericidal Properties**—Downes and Blunt in 1877 showed that the chemical rays could kill bacteria while D Arsonval and Charrain showed that they could kill the *Bacillus pyocyaneus*. In 1903  
 it was the middle third of  
 in which caused these bac

**Excitation**—Charcot in 1859 first expressed the opinion that it was the chemical and not the heat rays which produced sunburn and showed that the dermatitis caused by strong electric light was identical with that caused by the sun

In 1889 Widmark proved this definitely by using an electric arc

of rock-crystal and pieces of different coloured glass and also wrote his initials in Indian ink. He then exposed the arm to the rays from an 80 ampere arc for twenty minutes ten minutes at a distance of 50 metres and ten minutes at 75 metres. The result was that

first all parts were slightly influenced by the heat and then those parts which were unprotected or covered with rock crystal became red and inflamed and later desquamated and became pigmented while the parts covered by glass and Indian ink after the slight initial inflammation due to the heat did not further react. This experiment is interesting as showing that the effects due to heat appear at once and pass off quickly while those due to the chemical rays do not begin until after a lapse of three hours which agrees with the well known fact that a sunburn takes some time to develop.

Freund as the result of his experiments, concluded that the chemical rays penetrated into the skin. Bernard and Morgan found that the ultra violet rays were the active agent in producing sunburn.

The histology of solar erythema is not well known. Leredde and Pantrie made a biopsy on the skin of the shoulder of one of their friends who was suffering from sunburn of three days duration. The skin was in a condition of acute erythema without oedema or effusion. Under a low power of the microscope the epidermis appeared normal in thickness and disposition but the horny layer was exfoliated in places. The dermis was richer than normal in cellular elements and the connective tissue bundles were swollen. Under a higher power the intercellular spaces appeared larger than normal. The vessels of the dermis were dilated and there was a slight leucocytic infiltration while the connective-tissue cells were swollen. These appearances are exactly like those produced by the rays of an electric light.

If the sun's action stops at this the only change will be the deposit of the yellowish brown pigment in the skin so well known in the tropics. If however the action is more intense an exudation appears which may be sero fibrinous cellular or bloody while the depth to which these changes may extend depends upon the

- (1) Pigmentation
- (2) Vascular modification
- (3) Disease

(1) *Pigmentation*—It is well known that pigmentation follows sunburn but until recently it was not evident that it was of a protective nature.

In 1888 Wedding confirmed by Charcot first made this point clear though Unna in 1885 was the first to say that it was to be regarded as useful *masmoch* as it prevented the rays penetrating too deeply thus preventing inflammation.

In 1896 Finsen painted a black ring 2 inches wide round his arm

with Indian ink to imitate the colour of a negro's skin and then exposed it for three hours to a very hot sun. For a time the skin remained normal showing only a little redness at the edge of the

has — — — — —

| | | | |

This experiment of Finsen shows that the colour of native races

| |

against ultra violet rays

Dyson studied cutaneous pigmentation in 1911 and concluded that the formation of melanotic pigment was a normal function of the nuclei of the epidermis.

That the dark pigmentation is useful is shown also by the observation made by us on various occasions that natives suffering from

leucoderma under our notice. A native who had developed large leucodermic patches involving the whole of the face noticed that he could not see. The patient was examined and the patches were found to be on the face and the skin.

Generally speaking, the races of the world are as follows: West yellow, the race of the

become sunburnt in the tropics. On the contrary their skin, especially in localities where the climate is damp and hot, may take a peculiar whitish colour, even in cases in which the blood examination does not reveal any sensible decrease in the amount of hæmoglobin. As a result of an extended series of observations made by medical officers in the Philippine Islands, Chamberlain reports that the red cell counts averaged 5,200,000 per cubic millimetre in healthy American soldiers averaging twenty-six years of age after twenty months' service near sea level, which count does not differ from

in the muscles which is found in new comers in the tropics. This condition

speculative

On the other hand, there is most urgent need for protection from sunlight both as regards the eyes and the whole body. With regard to the effect of the tropical sun on man and animals, some most interesting experiments have been performed in the Philippine Islands by Aron and Gibbs separately. Rabbits and monkeys—i.e. animals with limited power of physical heat regulation—die if exposed to the sun's rays. The body temperature of these animals rises to fatal levels. However, the human body, with its more advanced heat regulation, is able to withstand the tropical sun for a certain period, but it is still necessary to take precautions to avoid sunburn and other harmful effects.

compensated by increased loss from the animal by such means as a strong wind, the animal suffers no discomfort. Insolation of the skull alone is without effect if the body temperature is kept within normal limits. Our own experiments showed that rabbits died in about an hour if exposed to the sun with their head shaved and lived if protected from these rays by means of red glass. Aron and Gibbs have also shown that if the human skin is exposed to the sun's rays the temperature of the area so exposed rises as a rule more rapidly and reaches a higher maximum in a dark skin than in a light, until the nerve endings of the latter are irritated by the prolonged exposure. The black skin is protective because it guards

number of sweat glands in the dark skin is also protective. The air

deposited in the tissues owing to unperfect oxidation due to heat and that of Darwin that it was due to a survival of those best fitted to withstand tropical disease—for he believed that pigmentation prevented the native from being attacked by the fatal miasmata of the country—cannot now be seriously considered.

As to the origin of the pigment, this question must be considered as far from settled. There are two possible sources for the melanin, viz —

- (a) The hæmoglobin of the blood
- (b) The cells of the epidermis

(a) *The Hæmoglobin*—This theory suggests that hæmoglobin is manufactured in the connective tissue and is carried to the skin by amœboid movement.

(b) *The Cells of the Epidermis*—This theory states that melanin is manufactured *in situ* by the epithelial cells and is not derived from hæmoglobin and is not carried to the skin by amœboid movement as above this

the diffuse melanin of the African negro skin is caused by melanin granules lying in and between the cells of the epidermis.

With regard to the pigmentation of different races it must be remembered that it is only absent in albinos and that it occurs in the epidermis of the areolæ and mammillæ of the breast the scrotum, labia majora and around the anus in white races, being contained chiefly in the large basal cells of the Malpighian layer, and to much less extent in the more superficial layers, and the connective tissue



cells of the papillary layer of the corium. In negroes the pigmentation is deeper and more diffusely spread in the epidermis.

(2) *Vascular* — It is said to be a p[er]manent condition of the skin and it is more marked in the tropics than in the Temperate Zone.

Light in general is believed to have an effect upon the blood which absorbs the violet and ultra violet rays and the red corpuscles under these influences probably absorb more oxygen.

(3) *Disease* — With regard to disease there appears to be no reason to doubt that the irritating effect of light has at least a part in the ætiology of Kaposi's disease (*Xeroderma pigmentosum*) which we have met with in the tropics where it would naturally develop rapidly in children prone to the disease.

There is also no doubt that these rays play a part in the production of the erythema of pellagra as will be described later on and they may have some effect in producing sunstroke.

Woodruff draws attention to the almost universal neurasthenia of white men in the tropics among whom he says insanity is more common than in Temperate Zones. In support of the latter statement he asserts that the insanity rate in the Philippine Army in 1901 was 2.02 per mille while in the United States from 1889-1898 it was 1.13 but he remarks that the diagnosis was not confirmed on the arrival of several of the Philippine soldiers in the United States as they recovered *en route*. He draws attention to the loss of memory which is very prevalent among the coast people of the tropics which is very useful in preventing this neurasthenia and in this he is also probably correct especially for ladies. Ordinary officials and business men however, cannot afford to rest in the middle of the day.

*Moon's Rays* — It is especially noticeable that the rays of the moon are subject to the same effects as the rays of the sun and considering the effects of its rays upon man.

Nevertheless in our own experience the rays of the full moon do produce headache and a certain amount of nervous irritability in persons who sleep in the open. The subject requires and deserves scientific study.

### ACCLIMATIZATION

When a person comes to the tropics he is not acclimatized to the condition of temperature, respiration, etc. as in the Temperate Zone—in other words becomes acclimatized. Further, if constantly

exposed to the sun's rays, his skin is apt to become more pigmented than normal, and to afford a certain degree of protection against its rays

tating influences of the tropical climate at an important growing period are not so fine physically as their parents

If however, the new-comer into the tropics desires to become acclimatized he must use some protection for his head and in many cases for his spine and eyes he must wear suitable clothing live in suitable houses, work in suitable offices, dwell in sanitary surroundings, avoid alcohol and exposure to the midday sun while roads in common use should be suitably constructed and well shaded

It is not within the province of this work to go into detail with regard to these matters, but a few brief remarks may be made

**Suitable Head-gear.**—All Europeans should wear a light helmet or topee covered with white or khaki-coloured cloth externally and ventilated This helmet should have a broad brim lined with green and prolonged down the back of the neck, should be lined internally with red, or red and yellow or black and should possess an internal band attached in such a manner that the head does not touch the frame of the hat, while it allows free circulation of the air

When persons are compelled to work under the tropical sun and to expose their backs to its rays, a thin strip of yellow and red coloured silk should be sewn into the shirt along the spine, or attached to the inner aspect of the coat by means of hooks and eyelets Eyes should be protected with dark glasses, or with

Ladies who are particular about the skin of the face should wear veils though these are very warm

The reasons of these requirements are while white reflects heat very well and absorbs very little and is therefore excellent externally yet it transmits the chemical rays while red and yellow absorb them

**Clothing**—Clothing should be loose and as light as possible in weight, and of a white or khaki colour externally Sambon some years ago devised a cloth called Solaro khaki coloured externally and red internally with the object of preventing the action of the sun's rays on the body It is made by using threads of yellow and blue twisted separately and together for the warp while red threads are used for the weft but these latter are brought back in the pro

**doublings** The general opinion at the present is however that white is by far the best colour

There are of the opinion that thin woollen

useful, howe

It should  
conductor o.

clothes Clothing should not be too heavy, nor too tight fitting Ladies should not wear too heavy skirts which congest the pelvic viscera

It is still to be worn by men at official  
unfortunate and may

ever be painted white

The amount of light  
d care should be taken  
m does not take place  
in the tropics is com-  
olling the light as are

tatties or tats made of grasses and wood and jalousies

Rooms should be capable of being cooled by punkabs or fans and should be lit with electric light whenever possible Ice-blocks are very useful in very warm weather or at meetings or in sick rooms The walls of rooms should be painted rose colour not white

Damp courses are required in the tropics as walls are apt to absorb moisture and become very damp Round roof tiles and

These are necessary to keep off the sun's rays  
ected from the glare of the sun  
trees with spreading branches—  
while plots of green grass are most valuable in towns There is not

muscular exertion should be done in the early morning or late afternoon and avoided in the middle of the day when a siesta is most beneficial. The absurd custom of making social calls at 12 noon in certain parts of the tropics should be discouraged, and a more reasonable hour substituted.

**Avoidance of Alcohol.**—Alcohol should never be taken before the sun goes down, for it unfits the individual for work, and is the most important predisposing cause of sunstroke.

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## CHAPTER IV

# TROPICAL FOODS

Preliminary Remarks—Evolution—Chemical composition—Quantity—Quality—Tropical food materials—Calculation of diets—Low protein dietaries—Vitamins—Lipoids—Little known matters—References

### PRELIMINARY REMARKS

It is not our purpose to attempt to give an account of the foods found in various tropical regions but merely to give a brief summary of some of the more important facts known to us with regard to tropical foods and their effects upon man.

The subject owes much to the labours of McCay in India while Wilson in Egypt has shown how this work can be extended to other parts of the tropics.

We will begin by tracing briefly the origins of foods as far as we know them.

### EVOLUTION.

At the present time human food is everywhere more or less cooked and it is

the world but to-day it is exceedingly difficult to trace the means by which this diffusion took place though it is probable that much of it was

... 334 B.C. and from Europe it was introduced into America

Beans were first cultivated by the Aryans but the haricot bean comes from South America while lentils are of prehistoric origin and spinach was apparently cultivated by the Medes and Persians Onions possibly originated in India while the

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The reason why man in Eastern countries eats so much rice must be sought for in the overpopulation of the Eastern Asia of long ago when the great difficulties began those large movements of peoples which we have noted in Chapter II

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these there are peoples who have begun to cultivate the ground while others still live upon the food which they can gather. Thus the Dravidian Mundas and Uraons eat insects lizards snakes rats jackals and pigs—or in other words anything which they can catch. Their daily dietary is composed of protein 80 grammes carbohydrates 500 grammes and fats 50 grammes which provides calories 2 800.

The Todas of the Nilghur Hills live upon milk the meat of buffaloes and of such animals as they can kill or capture. They take no vegetal food and hence like the carnivora require no salt which is an essential to vegetal feeders in order to prevent acidosis.

The Bushmen of South Africa and the Bedouin of Arabia are meat eaters.

The necessity for food is to provide heat and energy and to form new bodily tissues as well as to make good the wear and tear of existing tissues and to do this a community requires pure water and plenty of it and good and varied foodstuffs in quantity proportional to the numbers of the population a fact which the present war has made clear to nearly every family in the civilized regions of the earth.

Dietetics are based upon chemical and physiological considerations into which we will now inquire very briefly.

### CHEMICAL COMPOSITION

In order to meet the requirements of the body foods must be composed of the same essential chemical substances as that body. They are therefore made up of proteins carbohydrates fats mineral substances and water but these alone are insufficient to keep the body in health and they must be associated with vitamins and lipoids or nitrogenous fats.

Proteins may be obtained from the muscles bones and organs of animals used as food also from animal products such as milk and eggs while the many vegetal substances but particularly legumes nuts and cereals also provide this valuable food constituent. Their primary value is as tissue formers and their secondary value as heat energy producers but all proteins are not of equal value as we shall see later.

Carbohydrates are chiefly of vegetal origin and are principally

vegetal  
while the  
gh their

exact use is not known

Mineral Substances are compounds of sodium potassium calcium  
sulphuric  
oxalic and  
duce no heat  
and tissues  
of the total

weight

Water, forming some 58.5 per cent by weight of the human body is an indispensable

**Vitamines** are nitrogenous complexes which are essential for the growth or the well being of the organism

The food materials may therefore be classified into the great tissue forming protein aided by salts and water and the great heat energy producing carbohydrate and fats, aided by protein and essentials the work of which is not understood—viz vitamins and lipoids

As the heat and energy can be expressed in terms of the calorie (or large calorie written with a capital C) which is the amount of heat necessary to raise the temperature of 1 kilogramme of water one degree centigrade and as protein is the essential tissue former it follows that in calculations as to a diet two matters stand out—viz the quantity of protein in that diet and the number of calories which can be obtained therefrom

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ived in

Vitamines and lipoids we cannot measure and at present we are merely concerned with their presence

Therefore from a practical point of view the quantities which require calculation are the amount of protein in a diet and the number of Calories and this brings us to the subject of quantity

### QUANTITY

The only accurate method of determining the quantity of the various food factors of any given diet is by means of the *respiration calorimeter* in which the work done the heat generated and the

view of determining the quantity of protein carbohydrate and fat contained therein

With regard to the Calories produced by these various factors

and therefore 2.7 grammes of protein or of carbohydrate are *isodynamic* with 100 grammes of fat In other words 1 gramme of protein produces the same amount of heat as 1 gramme of carbohydrate—viz 4.1 Calories while 1 gramme of fat gives 9.3 Calories

It may perhaps be incidentally noted that 1 gramme of alcohol produces 7.0 Calories

The necessity for food new bodily tissues as existing tissues and to and plenty of it and good and varied foodstuffs in quantity proportional to the numbers of the population a fact which the present war has made clear to nearly every family in the civilized regions of the earth

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*Carbohydrates* are chiefly of vegetal origin and are principally of value as heat energy producers

*Fats* are

kingdoms

*Lipoids* or

exact use is not known

*Mineral Substances* are compounds of sodium potassium calcium magnesium manganese and iron either with carbonic sulphuric thacetic citric malic oxalic and fluorine. They produce no heat building up the fluids and tissues of the body in which they represent some 5 or 6 per cent of the total weight

*Water*, forming some 58.5 per cent by weight of the human body is an indispensable

Vitamins are a group of substances which are essential for the

classified into the great tissue forming protein aided by salts and water and the great heat energy producing carbohydrate and fats aided by protein and essentials the work of which is not understood—viz vitamins and lipoids

follows that in calculations as to a diet two matters stand out—viz the quantity of protein in that diet and the number of calories which can be obtained therefrom

Water has a food value of 1 in 1 000 and should be freely available in

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## QUANTITY

Then 1  
which in energy is the equivalent of 1 54 foot tons or in other words

view of determining the quantity of protein carbohydrate and fat contained therein

With regard to the Calories produced by these various factors,

and therefore 227 grammes of protein or of carbohydrate are isodynamic with 100 grammes of fat In other words 1 gramme of protein carbohydrate  
It may produce

Standard diets expressed in grammes have been determined for  
 ) kilo-  
 Tem  
 these

standards —

Observer	Protein	Carbohydrate	Fat	Calories
Ranke	100	240	100	1 324
Voit	118	500	56	3 055
Rubner	127	509	52	3 092
Moleschott	130	530	40	3 160
Atwater	125	400	125	3 315

below —

Atwater's Coefficients		Inter Allied Food Commission	
Ages	Mean Value	Ages	Mean Value
0 to 5	0.4	0 to 5	0.5
5 to 9	0.5	6 to 10	0.7
10 to 13	0.6	11 and over	
14 to 15		Males	1.0
Males	0.8	Females	0.83
Females	0.7		
16 and over			
Males	1.0		
Females	0.8		
All children, combined ages	0.51	All children combined ages	0.68

These figures may be compared with some Indian dietaries given by McCay —

Class of Person	Protein	Carbohydrates	Fats	Calories
Cultivators	52	475	25	2 350
Poor middle	50	400	50	2,310
Middle	70	300	90	2,350
Better	85-100	300-400	150	950-3 450
Bengal pri on d et	93	093	30	3,500

In Egypt, Wilson has inquired into the food of a Bedouin, and found that large quantities of rice and milk were used while meat

of a mixture of two parts of dura and one part of wheaten flour but on desert journeys was made entirely from wheat Onions and cheese were also used

He investigated the diets of two Egyptian men with the following results which however, are complicated by the fact that they are calculated from the amount used by the family —

Egyptian Men	Protein	Carbohydrate	Fat	Calories
Strong and healthy	69.1	628.0	47.5	3,326
Not very robust	82.0	520.0	49.0	2,870

Perhaps the best practical test is the British war ration which according to Lelean, was inadequate in the South African War producing loss of efficiency from neurasthenia and debility, as was afterwards proved by experimental marches performed upon it but which, in 1913, was altered to one containing a high amount of protein, which, indeed, is required to meet the needs of tissue repair It is as follows —

Protein	Carbohydrate	Fat	Calories
175	318	515	4,655

Work —

Nature of Work	Protein	Calories
Rest	100	2,700
Light work	112	3,000
Moderate work	125	3,500
Hard work	150	4,500

In Japan Oshima found that a jinrikisha man doing hard work consumes different foods during his periods of work and rest During work he eats large quantities of rice, and during rest quanti-

ties of fish, eggs, beef, and pork. He consumes about 20 to 30 ounces of beef or mutton per diem which works out at 158 grammes of protein and 5,050 Calories.

Light worked Japanese require 100 grammes of protein and about 3,000 Calories per diem.

In India McCay determined the amount of nitrogen excreted daily in the urine and adding to this the other metabolized nitrogen mentioned above, and converting this into terms of protein obtained the absorbed protein, which he calculated was 75 per cent. of the dietary protein.

For example, the urinary nitrogen being 6 grammes which is the equivalent of 35.5 grammes of absorbed protein, adding to this

words lives on a poorer protein supply than any other race investigated.

Further results of the experiment show that the matter of fact the reverse is true.

Our own experiences of Chittenden's dietary in the tropics is that the experiment if continued sufficiently long, lowers the resistance

as surprising as he

1 per kilogramme

This is certainly

of application to

temperate and cold climates where one would expect more food to be required.

metabolized per kilogramme

Voit, McCay's and Oshimura's

Bengalis and Oriyas (rice diet largely)	0.116-0.120
Chittenden	0.120-0.130
Beharis and Eastern Bengalis	0.140-0.160
Japanese poorer classes	0.177
Nepalese	0.180-0.250
Sikkim Bhutias	0.250
Average European	0.270
Thibetan and Bholan Bhutias	0.350
Nepalese Bhutias	0.420

Indeed McCay found that Indian dandy carriers, Indian rickshaw men, and Indian coolies performing exceedingly hard work, did so on a diet containing 175-200 grammes of protein and 6,300-6,500 Calories per diem whereas the British Army ration mentioned above only allows 175 grammes of protein and 4,855 Calories.

a diet

### QUALITY.

In this section we desire to know the amount of any given food which is absorbed and is therefore available for use in producing heat energy and repair.

The amount which is capable of being digested may be determined by artificial digestion of a sample but though said to give good results it is hardly reliable as to absorption.

A better method is to begin by giving the person to be experimented upon a dose of charcoal and after a few hours to administer a given quantity of the food to be tested of which the protein carbohydrate and fat factors are known. The fæces are in due course carefully collected and weighed and the total amount of nitrogen and of fat determined. From the nitrogen after deducting 0.5 grammes to allow for the daily amount of excreted metabolized nitrogen can be calculated the amount of protein in the fæces and this deducted from the amount of protein in the food gives the quantity absorbed.

The fat is determined in the same way by ether extraction while the carbohydrate is obtained by calculation.

Nature of Food	Protein	Carbohydrate	Fat
<i>Used diet</i>			
Total foods	9	97	95
Animal foods	97	98	95
Vegetal foods	84	97	90
Meat and fish	97	98	95
Eggs	97	98	95
Milk cheese etc	97	98	95
Cereals	85	98	90
Starches and sugars	—	98	—
Legumes (dried)	78	97	90
Vegetables	83	95	90
Fruits	85	90	90

Langworthy gives the table above showing the *coefficients of digestibility* or the amounts per cent of the foods which were digested. It must be remembered that by the word *digestibility* as used in dietetics is meant not *apparent digestibility* or the time a food requires to pass through the stomach but *actual digesti-*



bility which is the quantity of a given food which is absorbed

d

Nature of Work	Total Protein	Digestible Protein	Calories
Light	100	92	2 700
Moderate	115	105	3 300
Hard	175	160	5 500

It will be noted that the British Army ration is below Benedict's hard work calories by 745

So far we have been writing as though all proteins were of equal value but they are not as the researches of Rubner and Thomas have shown

The minimum quantity of protein required daily so that a man of average weight may live without drawing upon the proteins in his tissues is 30 grammes of animal protein—that is to say protein derived from meat and eggs

To reproduce this value 34 grammes of rice protein and 102 grammes of maize protein are required

This factor is called the biological value of protein and so far as we know has only been studied by its authors and by Wilson in Egypt as set forth below

100 Grammes of Food Material	Available Protein	Biological Value of Protein	Absorption Coefficient calculated on Available Protein
Meat	19.00	19.00	95
Wheaten bread	5.00	2.00	75
Millet bread	3.40	1.36	55
Rice	6.50	6.00	80
Lentils	19.30	10.70	70
Beans	18.60	10.30	70
Fresh vegetables	1.00	0.50	—
Dura	7.80	2.30	75
Millet	4.45	1.78	55
Milk (buffalo)	5.90	5.90	—
Dried dates	1.90	0.90	—
Fal Sudan (shell)	19.00	10.50	70

Wilson gives the following table of the diets in use at Egyptian prisons with suggested modifications —

Details	Avail- able Protein	Biologi- cal Value of Protein	Protein from Animal Sources	Fat	Avail- able Carbo- hydrate Gross less Five per Cent	Energy Value in Kilo Calories
<i>Hard labour diet</i>						
Before 1898	72.9	37.0	8.28	29.7	524.0	2,860
1898-1899	77.0	39.5	6.69	86.0	569.0	3,436.0
1900-1905	74.0	37.5	6.0	56.0	546.0	3,056.0
1905 (wheat bread)	96.0	57.8	22.5	46.0	547.0	3,058.0
1911 (millet bread)	83.0	53.34	7.5	46.0	521.0	2,937.0
Suggested	89.2	45.28	7.0	48.0	588.3	3,218.0
<i>Ordinary labour diet</i>						
1898-1899	68.5	34.5	6.0	56.5	529.5	2,978.5
1900-1905	57.1	25.4	2.5	48.0	400.0	2,501.0
1905 (wheat bread)	87.0	44.7	6.0	43.0	553.0	3,010.0
1911 (millet bread)	69.7	38.7	6.0	43.0	576.0	2,844.0
Suggested	78.175	40.95	3.0	48.0	527.0	2,861.0
<i>Non labour diet</i>						
1898-1899	49.0	21.5	—	39.5	404.0	2,165.0
1899-1905	42.8	20.0	—	28.5	345.0	1,890.0
1905 (wheat bread)	70.2	35.4	—	37.0	491.0	2,680.0
1911 (millet bread)	59.2	31.2	—	37.0	490.0	2,556.0
Suggested	65.20	31.9	—	36.0	450.5	2,442.0
Bread diet 1898-1911	38.0	16.0	—	12.0	375.0	1,803.0
Millet bread diet 1911	28.0	11.0	—	12.0	154.0	1,679.0

this factor in the following ratio —

Age in Years	Total Calories per Day		Fat in Grammes	Fat Calories per Cent of Total
	Gross	Net		
0 to 6	1,650	1,500	62	35
6 to 10	2,310	2,100	62	25
10 to 13	2,750	2,500	74	25
13 to 20				
Males	3,300	3,000	88	25
Females	2,750	2,500	4	25
Adult average bodily workers				
Males	3,300	3,000	70-88	20-25
Females	2,750	2,500	60-74	20-25
Adult sedentary workers				
Males	2,750	2,500	60-74	20-25
Females	2,200	2,000	47-60	20-25
Adult very heavy bodily workers	3,900 to 5,000		17-160	30

bility which is the quantity of a given food which is absorbed  
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<i>Nature of Work</i>	<i>Total Protein</i>	<i>Digestible Protein</i>	<i>Calories</i>
Light	100	92	2 700
Moderate	115	105	3 300
Hard	135	160	5 500

It will be noted that the British Army ration is below Benedict's hard work calories by 745

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The minimum quantity of protein required daily so that a man of average weight may live without drawing upon the proteins in his tissues is 30 grammes of animal protein—that is to say protein derived from meat and eggs

To reproduce this value 34 grammes of rice protein and 102 grammes of maize protein are required

This factor is called the *biological value of protein* and so far as we know has only been studied by its authors and by Wilson in Egypt as set forth below

<i>100 Grammes of Food Material</i>	<i>Available Protein</i>	<i>Biological Value of Protein</i>	<i>Absorption Coefficient calculated on Available Protein</i>
Meat	19 00	19 00	95
Wheaten bread	5 00	2 00	75
Millet bread	3 40	1 36	55
Rice	6 50	6 00	80
Lentils	19 30	10 70	70
Beans	18 60	10 30	70
Fresh vegetables	1 00	0 50	—
Dura	7 80	2 30	75
Millet	4 45	1 78	55
Milk (buffalo)	5 90	5 90	—
Dried dates	1 90	0 90	—
Fal Sudani (shell)	19 00	10 50	70

Wilson gives the following table of the diets in use at Egyptian prisons with suggested modifications —

(b) The Burma, Rangoon, or white rice, also variously described as 'uncured,' 'milled,' or 'polished' rice, which is prepared by milling the unhusked paddy until the husk, the pericarp and the surface layers of the

UNUSUAL FACTOR

Millet.—The sorghums, or millets, according to McCay show a very defect

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EXTRA

100 Grammes of—	Available Protein	Available Carbohydrate	Fat	Calories	Cost in Millimes
Millet bread	3.4	45.0	1.5	212.5	0.6577
Dura .. .. .	7.8	65.5	54.0	344.3	0.82
Soya bean meal..	32.0	28.5	2.1	269.0	0.80
Dried dates	1.9	47.0	0.6	253.0	2.44
Tal Sudani (shelled) ..	19.0	16.8	45.0	562.0	1.737

Chalmers Watson found by actual analysis that the daily dietaries of eight healthy English children between four and six years of age contained *protein 71 grammes fat 67 grammes carbohydrates 198 grammes* and yielded a total of 1725 Calories

A most important matter is that food must be made tasty and should be well cooked and look nice—matters of great importance in armies

### TROPICAL FOOD MATERIALS

Excluding the work of McCay and of Wilson there is very little information available as to the chemical composition the biological value or the absorptions of tropical food materials

over 400 000 000 individuals in the Japanese archipelago are very deficient in protein material and for this reason has to be consumed in large quantities in order that a sufficiency of this important factor may be

(b) The Burma, Rangoon, or white rice, also variously described as 'uncured,' 'milled,' or 'polished' rice, which is prepared by milling the unhusked paddy until the husk, the pericarp, and the surface layers of the

and well suited to its numerous uses as a food material, and provides a greater return in protein carbohydrates, and energy for the same outlay than any other cereal

Millet.—The sorghums, or millets, according to McCay, show a very defective protein absorption—e.g., *Sorghum vulgare* 53 per cent, and *Pennisetum*

into meal, from which unleavened bread, porridge or sauce is prepared, or they are meal

McCay considers that these dhals produce a great waste of nutriment in that 25.42 per cent of the

Tablet —

100 Grammes of—	Available Protein	Available Carbohydrate	Fat	Calories	Cost in Millesimes
Millet bread	3.4	45.0	1.5	212.5	0.6577
Dura	7.8	65.5	5.0	344.3	0.82
Soya bean meal	32.0	28.5	2.1	269.0	0.80
Dried dates	1.9	47.0	0.6	253.0	2.41
Fât Sudâni (shelled)	19.0	16.8	45.0	562.0	1.787

Chalmers Watson found by actual analysis that the daily dietaries of eight healthy English children between four and six years of age contained protein 71 grammes, fat 67 grammes carbohydrates 198 grammes, and yielded a total of 1,725 Calories

A most important matter is that food must be made tasty, and should be well cooked and look nice—matters of great importance in armies

As emphasized by Rho in making a dietary the racial food peculiarities, which are, after all, adaptations to climate, should always be taken into consideration—e.g., the beef eating British soldier requires a different dietary from that of the soldiers of Southern countries

### TROPICAL FOOD MATERIALS.

Excluding the work of McCay and of Wilson there is very little information available as to the chemical composition the biological value, or the absorptions of tropical food materials

protein and rich in starch forms the *patents* and the remaining part *household or bakers' flour*, while the flour from the whole wheat is called *Graham* and from the entire grain—*i.e.* with germ and semolina—is *standard*

Rice.—Turning now to the consideration of certain articles of food in

(b) The Burma Rangoon or white rice also variously described as uncured milled or polished rice which is prepared by milling the rice to remove the husk and the bran and the germ.

Millet.—The sorghums or millets according to McCay show a very selective protein absorption—e.g. *Sorghum vulgare* 53 per cent and *Pennisetum*

protein factor

McCay considers that these cereals produce a great waste of nutriment in that they contain a large amount of indigestible material. The following table shows the nutritive value of these cereals as determined by McCay.

extract —

100 Grammes of—	Available Protein	Available Carbohydrate	Fat	Calories	Cost in Millies
Millet bread	3.4	45.0	1.5	212.5	0.6577
Dura	7.8	65.5	5.0	314.3	0.8
Soya bean meal	32.0	28.5	2.1	269.0	0.80
Dried dates	1.9	4.0	0.6	253.0	2.14
Pat Sodani (shelled)	19.0	16.8	45.0	562.0	1.757



## CALCULATION OF DIETS.

In the calculation of diets it appears to us that two important matters must be considered—viz, the quantity of protein and the number of Calories

With regard to protein, having obtained from the tables of analysis the *available protein* in the food, it is necessary to determine its *biological value* and the quantity of the *absorbed protein*, and from this the Calories can be calculated. The fat and carbohydrate present no difficulties, except that it is necessary to know the quantity of the available matter which is absorbed. Having obtained the quantities absorbed, the matter is simple arithmetic

Protein fat carbohydrate 5 3 1

and that no diet should be based upon a minimum

Wilson has invited especial attention to the quantity of protein, which is not a producer of energy until the carbohydrate and fats are used up, but repairs wear and tear or is stored up as fat or carbohydrate

The nearer the quantity of protein is to the minimum the longer will the organism require to rebuild damaged tissue and the less will its powers of resistance be against disease. He states that

with more protein for the cool weather or cool regions

If this is not done then the increased heat must be got rid of by radiation and conduction, and this can be obtained by natural or artificial breezes or by exercise, when the extra heat of muscular

quoting

protein components of the diet, also it is necessary to consider the basis rather than on the nitrogen content. In determining the amount of protein the defective absorption of vegetable proteins must be allowed for.

in the tropics for drinking purposes should be 3 to 4 pints per head per diem

Salts are also of great importance and usually present in the food but with much vegetal food addition of ordinary table salt is necessary

### LOW PROTEIN DIETARIES

In tropical lands there are many peoples who live mainly upon cooked cultivated vegetal foods with but little admixture of animal foods and this is due to force of circumstances rather than to any desire of the peoples who would be glad enough to eat animal food if it could be obtained. Among these peoples the protein in the food sinks to a very low amount with as we shall presently see a

Food	Bengalis I	Bengalis II	Anglo Indians and Furians
Proteids in grammes	67.11	43.61	66.56
Carbohydrates in grammes	548.73	200.31	376.53
Fats in grammes	71.50	43.9	54.75

that it is the influence exerted by the food and particularly by the proteins of the food that is all important in determining the degree of ... powers of all manliness as at length important

results may be briefly mentioned

## CHAPTER V

# TROPICAL DISEASES

Preliminary remarks—Evolution—Geographical discovery—Endemicity—Epidemicity—Eugenics—Incidence—Distribution—Prevention—References

### PRELIMINARY REMARKS

BEFORE entering upon the systematic study of tropical diseases a few remarks may be made upon their evolution the influences of geographical discovery upon their dissemination their endemicity and epidemicity tropical eugenics the geography of tropical disease with special reference to countries and not to given diseases and finally the prevention of these diseases

It will be obvious that if these subjects were to be treated at all fully they would require several volumes each of which would be

he is surrounded and references are given whereby he may extend his reading and knowledge far beyond the confined limits of our manual

All tropical countries are inhabited by three different sets of

### EVOLUTION

The researches of the late highly talented Sir Armand Ruffer into *palæopathology* have demonstrated the existence of bilharzias & tuberculosis rheu

## QUARTAN FEVERS

before the chill begins varies at first from  $100.4^{\circ}$  to  $102.2^{\circ}$  but generally rises rapidly to its maximum—about  $104^{\circ}$  or  $105$

the cold stage he desired to wrap himself up with coverings he desires to throw these off. The skin feels hot and dry pulse and respirations increase the conjunctivæ become injected Vomiting and diarrhœa may take place and an erythematous sometimes appears. The temperature reaches its maximum soon declines.

This stage may last about three to four or more hours.

3 *The Sweating Stage*—Towards the end of the hot stage forehead is noticed to be damp and presently the sweat begins to appear profusely and great relief is felt by the patient. As sweating increases the temperature falls rapidly and the pulse declines. As the temperature approaches normal the patient falls into a sleep from which he will wake feeling much better with a normal or subnormal temperature. The total duration of the attack may be about eight to ten hours.

**THE INTERVAL**—After awaking from his sleep the patient is quite well though weak and generally goes about his ordinary duties during the two days of this interval. But signs are not wanting that everything is not well for the temperature is often subnormal and the pulse slow while in the blood the developing parasites are present.

to have a great tendency to relapse to go on for months and as it is said for years if not treated. The parasites rarely appear to multiply to any great extent in the blood and hence pernicious symptoms are usually absent. If left to itself the fever is supposed to gradually die out but to recur at times. Spontaneous cure however rare.

**Irregularities.**—Prolongation of the attack has been noted. Marchiafava and Bignami state that it may very exceptionally

may be very severe

man and animals as they did when hosts were first evolved only the matter is more complicated owing to the evolution of methods of defence upon the part of the host

In his most interesting and masterly book Adams has shown that a non pathogenic organism can be made pathogenic by injecting into an animal killed non pathogenic bacilli and then ten or fifteen days later the live bacilli. These when recovered from the tissues

words by means of a preliminary anaphylactic phenomenon a non pathogenic microbe may become pathogenic and a new disease be evolved

Similarly according to some of our observations when man is

the Sandwich Islanders in London long ago

Enough has been said to show the line of thought—viz the importance of environment the difficulty of impressing characters but the hereditary transmission of these characters when once evolved producing variation in the parasites while the reaction on the part of the host tending to produce an immunity against older

### GEOGRAPHICAL DISCOVERY.

Just above we have mentioned the introduction of pathogenic organisms to races to which they were previously unknown and as this is primarily due to geographical discovery we desire to invite the reader's attention to this factor in the dissemination of tropical disease as the discovery of new lands has eventually led to the betterment of means of communication and hence to the easier

and past wars spread to other regions where they were previously unknown and this brings us to the consideration of endemicity and epidemicity

### ENDEMICITY.

As so much epidemiological investigation of disease has of late been undertaken mainly due to the initiative of Manson and Sambon

But if a soil micro-organism, such as *Bacillus calvus*, has noted the presence of water and may be largely responsible for the formation of some of the oldest sedimentary rocks in much the same way as *Bacillus calvus* does its work to-day in the lagoons of Florida and on the Great Bahama Reef

When higher plants and animals evolved, the struggle for existence must have compelled minute animal and vegetal organisms to seek protection for themselves therein, and at first this protection would be used temporarily to tide over some difficulty

This being admitted, the parasite would desire an easy method of entrance into and escape from the body of the temporary host and hence the common infection of the alimentary canal with organisms

So long as the chemical substances produced by the metabolism of the parasite were innocuous or helpful to the host there would be no reaction on its part against the intruder and the two would live together in peace, as many bacteria do at the present day in the human alimentary canal and a condition of commensalism may be arrived at

is adaptation to environment—viz., to that portion of the host's environment in which is centred the invading organism

The parasite would naturally attempt to escape from the defensive chemical substances poured on to it by the host, and in so doing

of the kidney

All these changes of environment would cause variation in the protists (protozoan and bacterial), and if the same environmental conditions acted long enough, then these changes would become inherited (*vide* Adam), hence the origin of the numerous parasitic protists, animal and vegetal, and hence also the preservation of characters, including those complicated phenomena associated with the reaction of the host which we call the signs and symptoms of disease and the natural recovery therefrom. It appears to us that

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arising cer  
should no

be capable of developing enormously in a given district but there are still other factors to be considered and the first of these is the fact that the intermediary host if an animal depends for its existence upon the presence of a suitable food supply as well as suitable means for propagating its species

If anything untoward happens to these the intermediary host may die out but the problem is not quite so simple as this because the intermediary host itself may be preyed upon by some other animal or

If the being extinct its life-cycle another intermediary host or another entirely different method of leaving the human host or of being exterminated

The reduction in numbers of the intermediary host in a given area is one of the bases of prophylaxis in malarial fever yellow fever

Faced with these difficulties parasite can change its intermediate host and changes itself in so doing a plausible explanation of the slight effect of spirochaetes causing the relapsing fevers

relapsing fevers

On the other hand the malarial parasite would seem to be less capable of accommodating itself to a change of hosts for it would appear to be only capable of completing its life cycle in the less common anophelinæ and not in the more common culicidæ—a most

the effect of atmospheric study of the life cycle of the

malarial parasite has clearly shown that this has a marked effect upon the development of the oocyst in the anopheles It is also quite possible that other physical and chemical factors concerning which we are at present ignorant may play important parts in controlling the life of parasites

Finally the parasite itself may suffer from the attacks of another parasite a condition of affairs called hyperparasitism and thus becoming diseased may be unable to complete its life cycle and so become extinct

It will thus be seen that the problems connected with the appearance and the disappearance of a disease in a locality are extremely complex and that next to the parasite itself the most important factor is the intermediary host its food its life cycle and its habits—in one word its oecology

Hence in studying an endemic parasitic disease or a disease thought to be possibly parasitic it is not advisable to restrict one's

we feel that a few general remarks on endemicity and epidemicity as applied to disease in general may not be amiss

The presence of a given disease in a locality depends primarily upon the presence of conditions favourable for the action of the

tion—as for example the action of gravity in producing the trauma

example the traumatism due to ferocious animals which though occasionally taking place in other parts whether the animals have been artificially conveyed generally only occur in the natural habitats of these creatures

Chemical causes of disease have become largely cosmopolitan in distribution owing to improved methods of intercommunication and the manufacture of chemical principles still certain chemical causes have very restricted localities even to day because they are little known and only affect primitive peoples living in these regions—e.g. *Gloriosa superba* etc

The parasitic causes of disease may be animal or vegetal. The endemicity of a given parasite depends upon (a) the presence of non-immune human beings in which part of the life-cycle can be gone through and which for convenience we will term the *human hosts* (b) easy modes of escape from the human host into the exterior (c) suitable means of continuing the life cycle in the exterior— $\rightarrow$  in earth water air on or in other animals or plants *intermediary hosts* we will call them (d) ready means of re entry into the human host or into some other animal host in which the life-cycle is completed (e) partially immune animal hosts or partially immune human hosts to act as reservoirs or carriers to enable the parasite to complete its life-cycle without producing marked pathological changes in the host. Examples of these may be found in the malarial parasite attacking (a) the white man and the native child in West Africa and elsewhere and passing by the agency of (b) blood sucking into (c) *Anopheles costalis* which by the act of biting (d) conveys the parasite to another non immune host or to the partially immune adult native who acts as a reservoir or carrier

Another example is the passage of the plague bacillus from the rat via the flea back to the rat or man

Other examples are the amœbæ of dysentery which pass from man by the evacuations on to vegetal substances or into water by which they may be reintroduced into man direct or by the agency of flies

By a suitable non immune host is meant a host which while



## EUGENICS.

of  
for  
disease. In order to bring about an improvement two factors are open for consideration—viz, the parents and the child.

With regard to the parents, eugenics attempts to combat such racial poisons as are represented by alcohol and such social diseases

Alcoholism in the mother is a more serious matter than alcoholism in the father, because in the former the embryo is produced by

frequently the case is reared by such hand feeding as an alcoholic

at the present time any system of compulsory personal notification would fail to secure the advantages claimed. The Commission considered that undeclared venereal disease should be a ground for the annulment of a marriage. They further considered that advertisements of patent medicines should be prohibited, and apparently they were in favour of making it penal for unqualified

countries. At the same time, we desire to record our disapproval of attempts to combat these evils by the registration and general

researches solely to work in the hospital or the laboratory—for it must be remembered that the parasitic causes of some diseases are

completely detached endemic areas in order by carefully studying there in all the conditions of life and the habits of the human hosts to endeavour to find factors common to the different localities. A further study of these common factors from the point of view of possible modes of infection may indicate one or more possibilities and then these must be put to the crucial test of experiment with a view of ascertaining definitely the accuracy of the epidemiological observations.

### EPIDEMICITY

Chemical causes of disease may be spread from their original restricted area by modern methods of intercommunication—e.g. poisonous plants or their products may be brought from the tropics to the Temperate Zone and *vice versa*.

In order that a parasitic disease may spread from its endemic focus several factors are necessary—

1. These may be intermediary host

2. new area there

If these and perhaps other still unknown factors are present the disease will be able to spread with perhaps increased virulence first within the new area and then from one area to another until an epidemic or pandemic is produced. With the appearance of partial immunity in the human and animal hosts altered climatic and other conditions as well as the aestivation or hibernation of

3. conditions become too adverse for the life of the parasite or it dies when the epidemic dies away and the disease again becomes restricted.

and ever increasing subject of *Eugenics* to which we will now turn our attention.

produce lasting results, because, as we have already hinted, there is a belief that the third generation of children, with European parents, born and living entirely in the tropics, tend to degeneration in every way, and yet it is desirable that portions of the tropics should be permanently colonized from temperate climates if possible.

Lastly, the *infantile mortality* of most tropical countries is appallingly high, and this passes unheeded, even in places where a local dearth of labour indicates to those in authority the necessity for a

This is not a book on  
many years in various  
to recommend anyone

anxious to reduce the death-rate of a district to study the factors which contribute to the local infantile mortality, and to combat those of greatest importance, which will often be found to be tetanus and diarrhoea, the latter being associated with the question of a pure milk supply

Enough, perhaps too much, has now been written as regards lines of thought associated with the word eugenics as applied to the tropics, and we will now pass on to consider the incidence of disease in tropical countries

### INCIDENCE OF DISEASE.

In previous editions we gave in considerable detail the analysis of the causes of death in Ceylon and their incidence in the various

### DISTRIBUTION OF DISEASE.

#### Asia.

for years Typhus smallpox measles, whooping cough, and it is said, scarlet fever, occur there Bagdad sore is almost too well known to be mentioned, while beri beri occurred in the siege of Kut, and also among other troops

In *Arabia*, in addition to the fevers mentioned above ophthalmia is to be noted

Very little is accurately known as to the diseases of *Persia* though we possess a large manuscript written thereon many years ago Cutaneous Leishmaniasis, leprosy and some form of relapsing fever may be noted

As regards *India* in the Bombay Presidency the infantile mortality in native races is 220.08 in males and 219.07 in females per 1000 births In Bombay City the ratios were 557.24 and 569.66, but it is stated that these ratios are very fallacious

police supervision of prostitutes as we believe such a system is bound to increase rather than diminish the disease as it leads to

acted beneficially in bringing the seriousness of the subject home

ledging with honour the great work which New Zealand has done in Europe with regard to this matter. When a country with traditions like England has awakened from its sleep of ages a sleep largely due to its religious tendencies it is possible for tropical countries unhampered by these religious bands to do even more than what we see going on around us as we write these lines in London

either being better diagnosed or was increasing in many tropical lands

There ought to be no slums in tropical countries and where they exist it is the duty of the Government forthwith to formulate

lation and work questions but also in the diminution of tuberculosis

In the tropics there are two other eugenic problems to which we may invite attention and these are *food* and *half castes*

With regard to food the low protein dietary of the poorer native population of such regions as India is a great sociological problem being connected with religious sentiment. It leads to racial degeneration as we have already seen but its remedy is very difficult and must be done by a social movement on the part of the natives themselves

In the tropics the means of preventing the disease are

do not show this degeneration

This is a great discovery and if confirmed and acted upon may

The general death rate is causing some apprehension

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Europeans	57.32
Other nationalities	224.07
Malays	229.78
Indians	276.72
Chinese	302.03
Eurasians	307.69

The general death rate was 40.51 per 1,000 inhabitants and its factors were phthisis, beri-beri, unclassified fevers, dysentery, cholera, malaria, cancer, anaemia, sprue, dropsy, and parasites.

In Indo-China the enteric fevers, the diarrhoeas and dysenteries, liver and intestinal parasites, cholera, diphtheria, spreading ulcer, which used to be common, are still present.

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### Australasia

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The racial death rates are —

Europeans	1905	1906
Eurasians	14 36	14 41
Natives	20 89	26 99
	31 84	41 30

The year 1905 seems to have been unusually healthy

1896	
1897	2 086
1898	46 944
1899	86 191
1900	96 596
1901	33 196
1902	128 259
1903	184 752
1904	281 269
1905	223 957
	71 363

total

1 154 613

The population in 1901 was —

European (exclusive of cantonments)	18 804
Eurasian	6 557
Native	18 481 362

The above figures will thus give the reader some idea of the mortality caused by the plague in India

The next important cause of death is cholera —

1896	
1897	35 404
1898	57 109
1899	4 366
1900	8 579
1901	163 889
1902	13 600
1903	3 229
1904	1 825
1905	13 156
	5 396

The general term fever causes a mean of 310 420 deaths in the six years 1900-1905 most of this is supposed to be malaria

The simplest way to show the Bombay death rate is in the ratio per 1 000 inhabitants —

Cholera	2 11
Smallpox	6 20
Plague	9 18
Fevers	17 43
Dysentery and diarrhoea	4 90
Injuries	0 39
Other causes	12 12

Total

46 30

Respirator

Every tropical practitioner knows how much sickness and loss of work is due directly or indirectly to venereal disease and we feel that it is not too much in the interests of the employer and the employee alike to ask that the above should form part of the duties of every medical examiner of candidates destined for the tropics

in good order

**Alcohol**—The objections to alcoholic candidates are so well understood that the mere mention of this point is sufficient (see Chapters III V and LVI)

**Age**—We are convinced that no one under twenty one years of age or over fifty years should be allowed to proceed for service for the first time in the tropics We are also of the opinion that the nearer the minimal age is to twenty five years the better for

should also

visitors arrive

winter Th

tropics but the

Temperate Zone

**Energy**—The selected candidate should be warned that on first

his energy to outrun his common sense there will be trouble

*Moderation in all things* should be indicated as the motto for tropical life

**Duties of the Examiner**—We have insisted upon the medical examiner giving advice upon various points to the selected candi

the inner side of the sole of the foot in the Igmissail and Cairns districts which beginning as a small erythematous area spreads in the form of spirals and

Guinea  
parts  
as a  
prevalent  
in  
valvular heart disease rickets or gout locomotor ataxy or general paralysis  
while anemia was rare

#### Oceania

This is interesting for its diarrhoea dysentery and elephantiasis which have been studied in detail by Baker for its yaws leprosy and skin diseases which latter are common and of which tokelau and ringworm may be especially noted

#### Tropical America

The ... .. and in other  
activity of Tropical

Moreover it is in tropical America that the most brilliant prophylaxis with regard to malaria and yellow fever has been conducted



maintained unimpaired and the same holds good, but perhaps with more force for women who should be allowed leave to cooler tropical regions and to the Temperate Zone as often as circumstances permit.

When a woman returns to a temperate climate she should be advised as to the necessity of an immediate medical examination as well as of the dangers of the change into cooler weather.

### INVALIDING

benefit of his health

With regard to the first there are two quite different aspects of the case viz —

(a) **Invaliding for the Good of the Employer and Employee Alike** — This is a matter of common sense and is the kindest method of dealing with many cases.

(b) **Invaliding because of Health** — Temporary invaliding or sick leave should always be advised if the patient's health requires it, but his financial status should also be taken into consideration before too drastic recommendations are made.

More difficult by far is the question of permanent invaliding from service and in coming to a decision the medical practitioner has many points for consideration with reference to the present conditions.

considered

invaliding

On the other hand we are of the opinion that *mental symptoms* for permanent invaliding as the tropics

playing havoc with a patient in a tropical area which may not mean that he should be invalided from the tropics or from the service

### NATIVES

for

Schistosomiasis of any part of the body should cause rejection

date Our reasons for so doing are because he may be the only medical practitioner to examine the candidate before the tropics are reached

medical examination by the local medical officers immediately upon the arrival of the new official is advisable in order to see whether

symptoms to become serious owing to lack of knowledge

### WOMEN

European and American women proceeding to the tropics are usually either married or about to be married or because of the

should be specially tested as to their ability to stand quinine therapy and if this is found wanting should be educated up to a necessary quinine standard by regulated small doses before being allowed to begin their new life

Our experience makes us agree with Mrs Scharlieb that the medical examination of women should include the bony pelvis and

abnormalities are discovered the woman or her husband or both should be warned as to the possibilities of such abnormalities

At such an examination care should be taken to see that there is no obvious cause for dyspareunia as it may cause trouble to the woman and her husband This has been brought home to us in our long experience

counting

The necessity of having the urine examined on the occurrence of pregnancy should be impressed upon the wife and her husband as well as the requirements of diet exercise rest in the warmer hours of the day etc

White men as a rule require at regular intervals visits to temperate climates if their health strength and mental vigour are to be



and diffuse leucoderma if the candidate is to work in the sun but it is not possible to give further details which must be left to the common sense of the examining officer

The invaliding temporary or permanent of natives from a service particularly if a pension or a gratuity is available requires great

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## LIFE ASSURANCE

you get those lands which lie nearest to the Equator as the most dangerous because of the heat the endemic diseases the lack of

official mortality rate of 80 per thousand and an official invaliding rate of 95 per thousand but these had been reduced to 21.7 and 70.3 per thousand as long ago as 1903 and to day are probably much less

Very many tropical practitioners have had experience in the selection and rejection of tropical candidates for life insurance but unfortunately there are but few records to be found based upon this work

In 1897 and later Cantlie writing with regard to this matter stated +

perate climates

He states that the insurance companies deal with each case

residence in a proscribed area an extra premium should be paid but that after that period it should be reduced to one-half upon a certificate from the company's medical officer that no permanent

original sum and after thirteen years no extra premium should be charged provided always that the necessary certificate was

the first year or two after permanent abode in the Temperate Zone and he recommends that an extra premium be charged for these two years

The next paper published by Winter in 1909 we have been

climate every four or five years and that after years of residence  
per cent

history bad fam

(33 per cent) "

With regard to natives desiring insurance it is true that years  
but

and money lenders and petty merchants are bad lives & the best native risks are Government servants and native clerks in

if he does he

can than the same weight

our opinion

location with our groups of

and diffuse leucoderma if the candidate is to work in the sun but

care and the possibilities of malingering must be remembered. On the other hand the effects of such apparently harmless diseases as diffuse leucoderma should be borne in mind and injustice should, if possible, be avoided.

### LIFE ASSURANCE

dangerous because of the heat the endemic diseases the lack of sanitation and the imperfect food supplies.

But the advance of knowledge with regard to the prevention and treatment of tropical diseases and the dawn of tropical sanitation has reduced and is reducing the baneful effects of many of these factors. For example the West Coast of Africa used to have an official mortality rate of 80 per thousand and an official invaliding rate of 95 per thousand but these had been reduced to 21.7 and 76.3 per thousand as long ago as 1903 and to day are probably much less.

selection and rejection of tropical candidates for life insurance but unfortunately there are but few records to be found based upon this work.

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perate climates

He states that the insurance companies deal with each case individually and he suggests that during the first seven years of



proposers—viz the Western European the Southern European the Eastern and the Egyptian

In general he considers the first class to be a good risk on the same terms as in Europe but with regard to the second he considers that they should be examined in their own homes and each case should

excess and cigarette smoking are considered to balance the lack of alcoholic excess Rheumatic fever tuberculosis and syphilis are rife and he is liable to many tropical diseases including ankylostomiasis pellagra splenomegaly relapsing fever typhus fever and the schistosomiasis so that he is not a good risk until education and sanitation improve matters but Day says a proposer then in good health and whose children are successfully reared and whose education has been sound may be accepted for a whole life on European terms but this was contested during the discussion on his paper

The common sense of the whole matter is that there is an increased risk on the life of the European living in the tropics and that it is most marked during the earlier years of his residence there is a considerable risk in insuring native men and that this is greatly increased when considering native women

### EXPECTATION OF LIFE IN TROPICAL NATIVES

The expectation of life in India compared with the same for England may be gathered from the following table obtained from Hardy via McCay's book—

Age	India		England	
	Males	Females	Males	Females
0	24.6	25.5	43.7	47.2
5	37.1	36.1	52.7	51.9
10	35.5	34.4	49.0	51.1
15	32.3	31.7	44.5	46.5
20	29.2	29.3	40.3	42.4
25	26.3	27.0	36.3	38.5
35	21.1	22.4	28.9	31.2
45	16.5	17.9	22.1	24.0
55	12.2	13.2	15.7	17.2
65	8	8.7	10.3	11.3

The figures speak for themselves but they were compiled years ago and it is possible that more modern tables might show a better expectation of life



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PART II  
THE CAUSATION OF DISEASE IN  
THE TROPICS

PHYSICAL CAUSES  
CHEMICAL CAUSES  
PARASITES

## SECTION A

### PHYSICAL CAUSES

TEMPERATURE AND HUMIDITY

PRESSURE AND RADIATION

TRAUMATISM

## CHAPTER VII

# TEMPERATURE AND HUMIDITY

Preliminary—High atmospheric temperatures—Conclusions—References

### PRELIMINARY

HAVING finished with the introductory portion of our task we enter upon the second part of this book which is devoted to the *causation of disease* and is therefore essentially pathological. This subject is divided into three sections embracing the physical, chemical and parasitic causes of tropical diseases.

### HIGH ATMOSPHERIC TEMPERATURES

The present short chapter is confined to a brief consideration of the pathological effects of high atmospheric temperatures which are the most important physical causes of disease in the tropics and are only markedly evident when there is a definite amount of atmospheric humidity and therefore we have entitled this chapter *Temperature and Humidity*.

To exemplify what we mean we will quote a concrete example. In a certain tropical locality there were the usual high air temperatures with relatively low humidity and cool nights until about the middle of a month when the temperature rose to 116° F. the humidity increased, the sky became overcast and for twenty-four hours there was a brick-hot stroke—viz. the congestion of the meninges and brain and the marked congestion of the lungs which were almost black with stagnated blood. During this period there were a few deaths in the tropics.

temperatures and is named *heat syncope*.

We have investigated these two conditions experimentally. It was our practice in our journeys from Ceylon to Europe to test the effects of high air temperatures upon ourselves and such of our

fellow passengers as took an interest therein when passing through the Red Sea in the hot months thereof

The air of the engine-room in steamers is laden with aqueous vapour and certain corners can be found near the condensers with

month if a person lightly clad and in good health places himself

gradually rise and his pulse rate increase until temperatures of over  $102^{\circ}$  F are reached and the person breaks off the experiment because he feels uncomfortable after which his temperature slowly returns to normal

We have performed this simple experiment several times but on one occasion in a person who was not in very good health the temperature after reaching  $101^{\circ}$  F ceased to rise the skin became cooler the tension of the pulse altered remarkably and he began to look ill The experiment was quickly stopped and stimulants administered so that he did not suffer any serious effects though it was some hours before he felt quite right

These two experiments show that a high atmospheric temperature can act in two ways In the first there was a gradual rise of the body temperature which if continued long enough would probably have resulted in hyperpyrexia In the second after an initial rise the heart became embarrassed and if the heat had been continued there appears no reason to doubt that this person would have passed into a condition of syncope

and failure but no fever both of which are brought about by the same cause—viz high atmospheric temperatures associated with

historical account as follows —

High air temperatures whether in the day time or at night have been

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conditions the medical writers of ancient times were silent about them. This does not seem to be quite just for Paulus Ægineta, Oribasius and the Arabians certainly understood that there were head symptoms which they

system  
In 1869 Eulenberg and Vohl stated that the disease was due to the liberation of the gases in the blood and Weiskarl and Richardson attribute it to clotting of blood in the vessels.

In 1870 Valentinus stated that the disease was due to heating parts of the brain through the (2) as the

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Herrmann

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therefore was

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tissues without

( ) sunstroke  
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heat acting on



duced by the teeth and claws of species belonging to the families Felidæ and Ursidæ

In the Felidæ are *Felis leo* Linnæus whose habitat is Africa and Asia *F tigris* Linnæus habitat Asia *F pardus* Linnæus habitat India *F leopardus* habitat Africa *F onca* Linnæus habitat South America, *F pardalis* Linnæus habitat South America These

cats have a general dental formula  $\begin{matrix} 3 & \text{I} & 3 & \text{I} \\ 3 & \text{I} & 2 & \text{I} \end{matrix}$  giving thirty teeth

for the whole mouth

special teeth capable of

These animals feed on

recently slain but also on its body for a day or so after its death

*U malayanus* Their dental formula is  $\begin{matrix} 3 & \text{I} & 4 & 2 \\ 3 & \text{I} & 4 & 3 \end{matrix}$ —forty two

teeth in the mouth but these do not include carnassial teeth which added to the facts that they are not as a rule such foul feeders and often eat

from a se

however

of a septic nature

The importance of the septicity of these wounds has been well appreciated from the earliest times for in the fifth book of his

De Medicina Celsus remarks with regard to the bites of men apes dogs and ferocious animals "Omnis autem fere morsus habet quoddam virus" and on this he based his treatment

which have

gained when

lady by the

Helmstadt

in 1739

of either sex

als but the

persons most frequently injured are hunters (shukaris) and sports

## CHAPTER IX

### SOME TROPICAL TRAUMATISMS

animal agencies the larger  
 include the Proboscideæ the  
 due to physical agencies  
 —Powdered glass—Bamboo

#### REMARKS

IN the tropics many accidents and injuries occur which are but rarely met with in temperate climates. These accidents and injuries may be divided into two categories—traumatism brought about by animal agencies and those due to physical agencies—the bites of various animals and injuries from various objects from trees and lastly the peculiar deformity of the foot in high-class Chinese women brought about by bandaging the feet of young female children.

There are of course many other traumatism which might be

It will be observed that we make a distinction between the physical injury caused by the bite of one of the larger animals and the chemical injury caused by a venomous animal and we do this advisedly while fully recognizing that the former may introduce septic toxins sometimes in large amount as well as germs into the wounds so produced.

#### TRAUMATISMS BROUGHT ABOUT BY ANIMAL AGENCIES

Traumatism caused by the Larger Carnivora—The word traumatism is peculiarly suitable for this nomenclature as it is derived from the Greek words τραμα (a wound) and θηρον (a wild animal). The synonyms are Morsus and Rostro et Unguibus dilaceratio (Latin) δηγμα διεξ, διασρασμός κακώς σπαραγμός, (Greek) Morsures Déchirements musculaires Dilacérations Déplacement

must at first be guarded especially in Europeans as it is impossible to foretell how serious the intercurrent septic infection may prove to be

The treatment resolves itself into two distinct categories first the first aid when the rescue is effected and secondly the usual surgical treatment

With regard to the first aid the usual methods for arresting hæmorrhage combating shock and carrying the victim are too well known to require repetition but the thorough washing of the

and that antiseptics should be added to the water used to wash the wound

As a rule the hæmorrhage has more or less abated by the time the man reaches a hospital but any possible source of bleeding must be at once attended to and the wound thoroughly washed

free drainage as possible allowed Repeated and carefully applied antiseptic dressings must be carried out Antitetanic serum should always be administered with the view of preventing possible tetanus

As soon as there is any suspicion that cellulitis has supervened

left by the destruction of the tissues while massage and douches

At first the food should be of the lightest description—broths soups and milk diet—and later the ordinary hospital diets may be gradually introduced

With regard to prophylaxis the natives often adopt simple

knowledge of the habits of these animals. The most dangerous region to travel through is tall grass as the hunter or traveller and the wild beast may meet suddenly and it is here that an accident

especially dangerous animal under such circumstances is the tiger

come in mind, as the omission to remember this simple fact has often

caused by these bites and  
 m. . . . . With regard to the  
 for . . . . . nauled the victim if  
 able to stand, feels giddy turns pale becomes unable to stand  
 and, if the injury is not . . . . .  
 shock wit  
 and weak  
 his condit  
 remains  
 and suffe  
 these symptoms are

to  
 rate 110 to 120 beats per minute. Usually the torpor  
 continues for some time, but delirium may intervene. The mouth  
 and throat continue to be dry and thirst is still a marked symptom.  
 Locally the injured region may show rents and tears in the skin,  
 lacerations of the muscles tendons exposed and torn vessels and  
 nerves injured, and perhaps torn across with more or less hemor-  
 rhage. Bones may be exposed, bruised broken, or dislocated joints  
 may be exposed, opened and injured. Around the injured regions  
 the parts are bluish or red in colour and swollen and quickly

f  
 f  
 spreading or acute traumatic gangrene. Later, as the patient  
 recovers there is the liability of sinus formation and of stiffness in  
 joints.

It is very important to remember that malaria can occur as a  
 complication . . . . .  
 ture may a  
 regard to t  
 be obtained  
 condition, but the prognosis with regard to even slight wounds

but the symptoms signs and treatment resemble those already mentioned under the heading of traumatism caused by the *Canis* and need not be repeated

Among the Bovidae the buffalo is very dangerous— *g Buffelus indicus* in India and Ceylon and *Bubalus brachyceros* in Central Africa—the danger being deep penetrating wounds of the limbs chest or abdomen as well as severe punctured lacerated and contused wounds in any part of the body

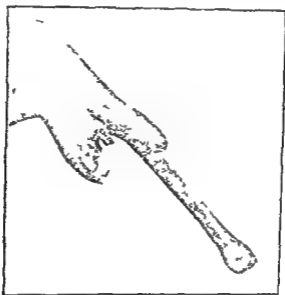


FIG 8—CAMEL BITE

(From a photograph given to us by Christopherson)

Here attention may be drawn to the fact that horses may become affected with hydrophobia which in animals treated as family

Kicks may produce serious contusions and wounds as well as injuries to internal organs

The Rhinoceros—*Rh indicus* *Rh javanicus* *Rh siamensis* *Rh africanus*

*I am  
when  
out or  
s into  
h will  
fe out*

of the ferocious creatures which he is hunting and especially to know what they usually do when wounded. In travelling in the African bush a zareba with a fire is usually a good protection during the night.

**Traumatism caused by the Smaller Carnivora**—Under this heading come the traumatism caused by members of the family Canidæ of which *Canis lupus* Linnæus the wolf *C aureus* the jackal and *C familiaris* the dog may be quoted. Their general dental formula is  $\frac{3 \text{ I } 4}{3 \text{ I } 4}$  forty two teeth. A wolf bite resembles

that of the larger Carnivora while jackals generally attack children inflicting severe wounds often of a septic nature. The

every now and again and are compelled to seek protection against possible infection by treatment at one of the now numerous Pasteur Institutes. In our opinion some attempt should be made by muzzling dogs for a period of at least two years throughout a country and the destruction of all dogs not so protected to diminish this danger. We also consider that the destruction of all ownerless dogs—i.e. dogs without a collar on which a name is engraved—should be conducted by a systematic organization.

The parasite of hydrophobia is described in Chapter XXII p 535

One of the most important forms in which rabies attacks dogs is that called dumb rabies in which the lower jaw early becomes paralyzed this is a form often overlooked for some time by non medical people and is a source of great danger.

Anyone interested in this subject must however consult a book of general medicine as this is hardly the place for a description of rabies.

Rat and cat bite diseases are described in Chapter LII p 1356

**Traumatism caused by the Ungulata**—Among the Herbivora there are two families which are celebrated for vicious attacks upon man and these are the Camelidæ and the Bovidæ.

*Camelus bactrianus* Linnæus may at times have a bad temper and it is often dangerous for a stranger to approach a camel for its bite is as a rule a serious injury the deep tissues being crushed and lacerated while the bones may be crushed broken or dislocated and tendons bursæ and joints may be lacerated or opened and last and by no means least because of the possibility of gangrene and severe septic infection. The bites generally occur on the upper or lower limb but are also well known on the head and face. The shock from such severe injuries is naturally very great.



The Suidæ or pigs are commonly met with in tropical jungles and are of importance because of the way in which they eat the remains of persons who have been lost in the jungle. All the soft parts are completely destroyed while the bones are broken so that it is impossible to say whether the unfortunate people died or were killed whether they met with an accident or simply lost their way whether the pigs ate them while dying or only after death.

Severe lacerated wounds can be inflicted by the tusks of an enraged boar.

**Proboscidea**—The elephants—*E. africana* and *E. indicus*—usually kill their victim by stamping upon him until the soft parts are terribly crushed lacerated and bruised while bones are broken. They also seize people by means of their trunks and dash them against surrounding objects on the ground.

**Traumatism caused by the Larger Reptilia**—The crocodiles proper in the tropics have the names crocodile, alligator, gator, and caiman were synonymous.

but this is by no means so. Some twelve species of crocodile are well known. They possess a most formidable array of teeth expressed as a rule by the formula  $\frac{18}{15}$  of which the third and ninth in the upper jaw are longer than the others and are respectively lodged between the second and third and the eighth and ninth teeth in the lower jaw in which the first fourth and eleventh teeth are the strongest. The muzzle of the crocodile is longer than that of the alligator. The muzzle of the alligator is relatively short and broad.

The muzzle of the gaviel is very long. The two species of importance are *Gavialis schlegelii* of Java and Borneo and *G. gangeticus* of the Ganges. The latter has a dental formula  $\frac{28}{25} \frac{29}{26}$  teeth.

The following list modified from Ditmar gives the names and habitats of these formidable reptiles—

- A Snout extremely long and slender  
*Gavialis gangeticus* India *Goitoma schlegelii* Borneo and Sumatra
- B Snout very sharp slender and triangular—  
*Crocodylus cataphractus* West Africa C *johnstoni* Australia C *intermedius* Orinoco
- C Snout bluntly triangular—  
C *porosus* India and Malaya  
C *moreletii* Guatemala and Honduras
- C *moliticus* Africa
- C *thomasi* Cuba



## POISONS

### I CRIMINAL POISONING

- 1 Homicide
- 2 Suicide
- 3 Infanticide
- 4 Abortion
- 5 Robbery
- 6 Pseudotherapy
- 7 Mimicry
- 8 Aphrodisiac

### II ACCIDENTAL POISONING

### III STIMULANT AND SEDATIVE POISONING

### IV ORDEAL POISONING

mortal wounds though they seldom were able to get away with

Usually the patient is in a state of extreme prostration covered with a cold sweat and having a hardly perceptible pulse

More rarely there are only a few triangular or irregular lacerated wounds showing that the shark did not obtain a proper hold of the victim

hours  
appear  
course

Man — The bites inflicted by the Kru men on the West Coast of Africa in quibrels are or were of not uncommon occurrence and it was also fairly common to meet with injuries on the knuckles caused by scratches from their teeth

These wounds were usually considered to be serious for although the teeth of the African appear to be in excellent condition of repair and cleanliness still the slightest scratch may lead to exceedingly severe inflammations as the present writers well know The most careful antiseptic treatment must be applied at once

### TRAUMATISMS DUE TO PHYSICAL AGENCIES

Foot-binding — The bandaging of the feet of young female children

valgoid position

On examining one of these feet it is noted that it is very short and

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pres  
normal foot

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Tl  
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of the metatarsals are approximated towards the os calcis 1110

We leave to future chapters the consideration of poisons used in war fishing hunting and trade as well as poisonous food and the effects of venomous animals

## I CRIMINAL POISONING

Criminal poisoning has been in existence from the earliest and most primitive ages and as it requires a highly and specially trained chemist with a well equipped and up to-date laboratory to detect many of the tropical vegetal poisons the reader will not be surprised to note that criminal poisoning was formerly almost unknown in the

that in Africa only the fetish man knows how to present virulent poisons in acceptable forms to his victims whereas persons with an elementary knowledge of poisons are common in the East

The poisons of different tropical countries vary according to custom and tradition and also according to the plants which happen to grow in the vicinity It is said that arsenic in the East

attributable to arsenic were found by advanced chemical research under an exceedingly able chemist to be due to *Cerbera odollam*

We are therefore of the opinion that every tropical country requires a well equipped toxicological laboratory with well trained research chemists whose business *inter alia* should be to make themselves acquainted with the poisons of the land in which they are residing with the view to discovering tests whereby these poisons may be detected and so justice done in criminal cases This is

the first of the most important methods of bringing the use

into

### Inorganic Poisons

The most common poison is *arsenic* in some form and the next is *perchloride of mercury* followed closely by the *mineral acids* Salts of copper have been used for homicidal purposes but generally poisoning from this source is accidental and due to the use of *copper* cooking utensils while the preparations of antimony are seldom used

### Organic Poisons

These may be derived from animal or vegetal sources but the latter are far more *en evidence* and are largely derived from plants

## CHAPTER X

### POISONS

Preliminary — Definition — Classification — Criminal poisoning — Accidental poisoning — Stimulant and sedative poisoning — Poisons used in trial by ordeal — References

#### PRELIMINARY

THE chemical causes of tropical disease or the tropical intoxications are numerous and it is impossible in a book of this size to do

Poisons and Venomous Animals and as we begin the study of these matters in the present chapter it is as well to state what we mean by a poison

#### DEFINITION

A poison is any chemical substance which when introduced in sufficient quantity into or brought in contact with the living organism is capable of producing a variation in the structure the chemical composition or the functions of the whole or any part of that organism which exceeds the limits of physiological variation

Such a definition includes a large number of substances and therefore some arrangement is necessary in order that they may be considered systematically

#### CLASSIFICATION

Poisons have been classified into mineral, vegetable and animal

- I Criminal Poisoning
- II Accidental Poisoning
- III Stimulant and Sedative Poisoning
- IV Trial by Ordeal

Country	Natural Order	Genus and Species
India and Ceylon	<i>Lamiaceae</i> De Candolle 1816	<i>Aconitum</i> Tournefort with <i>A. ferox</i> Wall and <i>A. napellus</i> Linnæus in the Himalayas <i>A. luridum</i> Aorte in Sikkim and <i>A. lycoclonum</i> Linnæus in Kashmir
	<i>Ipocynaceae</i> Lindley 1836	<i>Cerbera</i> Linnæus 1753 with <i>C. odollam</i> Gaertner and <i>C. thevela</i> Linnæus <i>Verum</i> Linnæus has <i>N. odorum</i> Solander 1729 <i>Urechites</i> is represented by <i>U. suberecta</i> Mucker
	<i>Liliaceae</i> Linnæus 1751	<i>Gloriosa</i> Linnæus with species <i>G. superba</i> Linnæus
	<i>Euphorbiaceae</i> A de Jussieu 1824	<i>Jatropha</i> Kunth with one species <i>J. curcas</i> Linnæus 1753
	<i>Loganiaceae</i> Auctores	<i>Strychnos</i> Linnæus with <i>S. nuxvomica</i> Linnæus <i>S. ignata</i> , Bergmann <i>S. colubrina</i> Linnæus
Dutch Indies	<i>Leguminosae</i> de Jussieu 1789	<i>Vitellia</i> Wight and Arnott with <i>M. sericea</i> Wight and Arnott
Pacific Islands	<i>Myrtaceae</i> R Brown 1811	<i>Barringtonia</i> Forster with <i>B. speciosa</i> Linnæus and <i>B. rarotongae</i>
	<i>Apocynaceae</i> Lindley 1836	<i>Cerbera</i> Linnæus with <i>C. lactaria</i> Hamiltou
Brazil	<i>Sapindaceae</i> de Jussieu 1811	<i>Laullima</i> Linnæus with <i>P. pinnata</i> Linnæus
	<i>Euphorbiaceae</i> A de Jussieu 1824	<i>Hura</i> Linnæus with <i>H. crepitans</i> Linnæus
	<i>Loganiaceae</i> Auctores	<i>Spigelia</i> Linnæus with <i>S. anthelmia</i> Linnæus <i>Thevetia</i> Linnæus with <i>T. ahova</i> Linnæus
British Guiana	<i>Sapindaceae</i> de Jussieu 1811	<i>Melicocca</i> Linnæus 1763 with species not mentioned
West Africa	<i>Solanaceae</i> Lindley 1836	<i>Hyoscyamus</i> Tournefort with <i>H. falsus</i> Cosson
	<i>Iridaceae</i> Lindley 1836	<i>Moraea</i> Linnæus with <i>M. collina</i> Waldtulpe
	<i>Cactaceae</i> Lindley 1836	The Oro of Sierra Leone
South Africa	<i>Iridaceae</i> Lindley 1836	<i>Moraea</i> Linnæus with <i>M. collina</i> Waldtulpe
	<i>Zygophyllaceae</i> Lindley 1836	<i>Melanthus</i> Linnæus with <i>M. major</i> Linnæus and other species

and vomiting muscular and cardiac weakness and finally death takes place from failure of the heart or respiration

*Barringtonia* Forster 1776 (*Myrtaceae*) with the species *B. speciosa* Linnæus and *B. rarotongae* is said to be used for criminal poisoning in Oceania

- 1 Poisons used for homicidal purposes
- 2 Poisons used for suicidal purposes
- 3 Poisons used for purposes of infanticide
- 4 Poisons used for procuring abortion
- 5 Poisons used with the intent to rob
- 6 Poisons used with the intent to cure disease
- 7 Poisons used with the intent to simulate injuries
- 8 Aphrodisiacs

### 1 Homicide

Animal and vegetal substances are used for this purpose but of the former the best known is *viperine venom* which is said to cause gastritis gastro intestinal hæmorrhages and even death. It is known to Ceylon natives that the venom of *Vipera russelli* Shaw locally called the *tic polonga* is supposed to be poisonous when administered by the mouth and we were informed that this was

which the recent venom administered orally by means of toddy to a monkey proving a complete failure we were left in doubt as to the possibility of such poisoning

The venom of the Colubridæ is harmless when taken by the mouth provided that there are no cracks or abrasions. Its virulence is destroyed by saliva and by pancreatic juice.

Vegetal substances are sufficiently numerous but unfortunately, as far as we know, few are on record. The table given on p 164 is an imperfect list.

With reference to the table a few notes arranged in alphabetical

that in India  
poison which  
s) *A. napellus*

L (Himalayas) *A. luridum* Aorte (سلكوم) *A. lycotonum* L (Kashmir), whilst *A. heterophyllum* Wall and *A. palmatum* Don are said to be less poisonous.

The root is the principal source of the poison, which however also exists in the leaves and stem.

Aconite is used for homicidal purposes as a cattle poison and may at times be taken accidentally. The fatal dose is said to be 30 grains of the root and the fatal period usually three to six hours.

The symptoms are tingling of the lips tongue mouth and fauces, followed by numbness and anaesthesia, burning pains in the stomach

1 2 3 4 5

Death may take place in four hours. The post mortem reveals congestion of the brain and membranes with extravasations of blood congestion of the lungs liver and kidneys and inflammation of the mucous membrane of the stomach.

The treatment is that for irritant poisoning in general.

*Hyoscyamus falezlex*—The Tuaregs are said to use *H falezlex* to kill travellers.

therefore the action of symptoms which may vomiting spasms with with short intervals of



FIG. 10.—*Gloriosa superba* LINNÆUS

*Jatropha curcas*—*J. curcas* Linnæus (Euphorbiaceæ) the jura tree of India has a seed which is called the physic nut from which the oil can be expressed which is an irritant to the skin and a purgative.

The symptoms of poisoning are vomiting purging abdominal pain derangements of the special senses muscular twitchings and loss of memory. The treatment after getting rid of as much of the poison as possible is lime juice and stimulants.

*Melanthus* (Melianthaceæ)—Various species of *Melanthus* are said to be very poisonous by Grey who suspects the possibility of their use by South African Bushmen.

*Melicocca* (Sapindaceæ) occurs in British Guiana where it is called kinnup. In a two-year old child it caused convulsive twitching.

*Cerbera odollam*—*C. odollam* Gaertner (Apocynaceæ) is a very common plant in Ceylon and is similar to *C. theceta*. Its correct name is probably *C. manghas* Linnaeus 1753. Its chemical properties have not yet been properly worked out and it is said that it

is used for arsenical poisoning. The treatment is the same as for irritant poisoning in general.

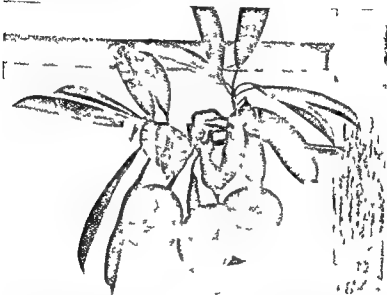


FIG. 9.—*Cerbera odollam* GAERTNER

*Cerbera theceta* (*theceta*) is a yellow oleander of India is a shrub about 6 to 12 feet in height with yellow bell-shaped flowers and globular green fruit. It is highly poisonous, its action being due to a milky juice.

three seed  
symptoms  
and pain in

the throat. The pulse is very soft and slow, thirty to forty beats a minute which Windsor says is characteristic. Later it becomes weak, very rapid and irregular. Death results from cardiac failure. The treatment is the same as for ordinary irritant poisoning.

In  
pu



identical with, the scillitine of squills and therefore the action of *C. foetida* is not unlike that

may take place in some months

The post mortem reveals congestion of the brain and membranes, with extravasations of blood, congestion of the lungs, liver, and kidneys and inflammation of the mucous membrane of the stomach

The treatment is that for irritant poisoning in general

*Hyoscyamus falezlez*—The Tuaregs are said to use *H. falezlez* to kill travellers



FIG. 10.—*Gloriosa superba* LINNÆUS

*Jatropha curcas*—*J. curcas* LINNÆUS (Euphorbiaceæ) the jura-tree of India has a seed which is called the 'physic nut,' from which the oil can be expressed which is an irritant to the skin and a purgative

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of the hands and feet fixed gaze vivid flush on skin distension of abdomen suppression of urine rise of temperature to  $101^{\circ}\text{F}$ . The poisoning was said to resemble that produced by belladonna. Other species are said to stupefy fish and also to be used as arrow poisons.

poisonous

*Milletia sericea* (Leguminosae) is a poison of the Dutch Indies causing severe diarrhoea collapse and death.

*Morea collina*—*M. collina* (Indaceae) the wild tulip of South Africa is said by Grey to have been used by a Bushwoman to poison a number of people two of whom died.

The symptoms were severe vomiting and a feeling of constriction across the chest feeble and intermitting heart and a tendency to coma for hours before death. The post mortem showed no rigor and no inflammation of the stomach. The heart musculature was flaccid and the right side full of blood.

*Nerium odoratum* synonym *N. indicum* Mill 1768 (Apocynaceae) is the white oleander and grows commonly in India where it is a well known

poison. It is rarely used for homicidal purposes but more generally for suicide abortion and accidental poisoning.

The root is the portion used but all parts are poisonous. The active principles are neriodorin a powerful cardiac poison acting something like digitalis karabin a cardiac poison with also a strychnine-like action on the spinal cord.



FIG. 11.—*Nerium odoratum* = SOLANDER

times lockjaw

Towards the end the patient becomes drowsy then insensible,

and finally dies from cardiac failure. The post mortem reveals

negro slaves in Brazil to poison their masters and to depend upon an alkaloid timber for its action. Adverting to our remarks on *Melicocca* we may note that the seeds of *P. sorbilis* are the source of the Guarana bread of Brazilian aborigines used when travelling and as a remedy being said to be a stomachic febrifuge and aphrodisiac.

*Sablier crepitans* Linnæus also named *Hura crepitans* (Euphorbiaceæ) grows in the Antilles and has been imported into tropical Africa. Its fruit is purgative and is believed to be used frequently for criminal purposes being said to produce violent vomiting and purging with tenesmus constriction of the throat and syncope. Ruiz however has thrown doubt on the possibility of its really being the cause of these criminal poisonings. It is to be noted that the same symptoms are produced by *Croton tiglium* Linnæus (Euphorbiaceæ) which is found in India Cochin China tropical America and Africa.

*Spigelia anthelmia* Linnæus (Loganiaceæ) has the reputation of being very poisonous and has been used criminally in tropical America in Brazil and the Antilles where it is still used as an anthelmintic. It is said to produce somnolence convulsions and death.

*Strychnos*—*Strychnos* (Loganiacæ) is a genus with several poisonous species among which may be mentioned—

*Strychnos nuxvomica* Linnæus

*Strychnos ignata* Bergmann

*Strychnos colubrina* Linnæus

Poisoning by the alkaloid strychnine is frequently met with in India but is usually accidental. The symptoms of strychnine poisoning are bitter taste in the mouth tetanic spasms opisthotonus risus sardonicus and death from asphyxia or collapse. There are no characteristic post mortem appearances except congestion of the brain and spinal cord. The treatment is to empty the stomach by emetics and to administer chloroform or chloral hydrate and when asphyxia threatens to perform artificial respiration.

*Thevetia ahoval*—*T. ahoval* (Apocynaceæ) is allied to the oleanders mentioned above and has an alkaloid thevetosin said to cause gastric irritation and difficulty of breathing.

of the hands and feet fixed gaze vivid flush on skin distension of abdomen suppression of urine rise of temperature to  $101^{\circ}\text{F}$ . The poisoning was said to resemble that produced by belladonna. Other species are said to stupefy fish and also to be used as arrow poisons.

We may perhaps mention here that in this order are many excellent and edible fruits thus *Melicocca bijuga* a West Indian tree is cultivated in Brazil because of its agreeable slightly acid berries but on the other hand the leaves and branches of other species are poisonous.

*Milletia sericea* (Leguminosæ) is a poison of the Dutch Indies causing severe diarrhoea collapse and death.

*Morea collina*—*M. collina* (Indicæ) the wild tulip of South Africa is said by Grey to have been used by a Bushwoman to poison a number of people two of whom died.

The symptoms were severe vomiting and a feeling of constriction across the chest feeble and intermitting heart and a tendency to coma for hours before death. The post mortem showed no rigor and no inflammation of the stomach. The heart musculature was flaccid and the right side full of blood.

*Nerium odoratum* synonym *N. indicum* Mill 1768 (Apocynaceæ) is the white oleander and grows commonly in India where it is a well known

poison. It is rarely used for homicidal purposes but more generally for suicide abortion and accidental poisoning.

The root is the portion used but all parts are poisonous. The active principles are neriodorin a powerful cardiac poison acting something like digitalis karabin a cardiac poison with also a strychnine-like action on the spinal cord.

The symptoms are therefore (1) those of gastric irritation—viz vomiting pain in the stomach frothing at the mouth from salivation but as a rule without diarrhoea (2) cardiac symptoms producing at first a slow pulse which finally becomes quick and weak—the respirations are rapid from the first (3) strychnine-like symptoms of twitching of the muscles tetanic spasms with cramps and trismus lockjaw.

Towards the end the patient becomes drowsy then insensible



FIG. 11. *Nerium odoratum* SOLANDER



*Urechite suberecta* (Apocynaceæ) is the Savannah flower of Jamaica and other West Indian Islands which was so celebrated in the days of the 'Oberon Man' and about which so many tales were told. The truth is that there are two glucosides, *urechitin* and

or so

## 2 Suicide

Of all the poisons used for suicidal purposes, opium is by far the most usual though *Nerium odoratum*, *Cerbera odollam* and *Gloriosa superba* are also at times employed, the former especially by women in India. Rarer poisons are *Calotropis procera*, *Cerbera thevetia*,aconite, prussic acid and veratrine (meeta bishi). The action of all these poisons is described either in ordinary works in toxicology or has already been mentioned.

## 3 Infanticide

Infanticide exists in the tropics in two forms, the first irrespective of sex and said to be due in India (Waddell) to the high caste Hindu prohibiting remarriage of widows and secondly female infanticides to prevent too many daughters growing up. A few

with the  
of opium  
(native  
nurses) are apt to soothe a baby to sleep by dipping the finger in  
Laudanum and giving it to the baby to suck. Such treatment is  
highly deleterious to the child and the intensely contracted pupils  
should make the practitioner suspect its use in an obscure case of  
illness.

*Calotropis gigantea* Robert Brown and *C. procera* Robert Brown (Asclepiadaceæ) called mador in Hindustani and erukam in Tamil have been used in India for infanticide and abortion rarely for suicide and more rarely for homicide. The symptoms are vomiting, profuse salivation, severe tetanic spasms, extremely slow and stertorous breathing and dilatation of the pupils. The active principle is a yellow bitter resin but there is no alkaloid. The treatment is the same as for irritant poisoning.

*Tetradlea* is also used as an infant poison.

## 4 Abortion

Criminal abortion is very common in the tropics. In India it is said to be common among Hindu women because they are not allowed to remarry.



*Datura fastuosa* (Solanaceæ)—Poisoning by datura is common in India. The seeds are usually those of a cerebral coma but in both delirium is peculiar



FIG 12—*Datura fastuosa* LINNÆUS

for example the people affected may be found searching their bedding most vigorously for some lost article. When death occurs it is due to cardiac failure. The fatal dose is not actually known but may be about 10 to 15 grains of the seeds. Waddell puts the mortality at about 18½ per cent. The post mortem characteristics are wide dilatation of the pupils, congestion of the brain meninges and the lungs and other viscera.

#### 6 Poisons used with Intent to Cure Disease

Decoctions of bark *Trianthema pentandra* Linnæus are used in the Sudan for the purpose of curing gonorrhœa unfortunately sometimes this remedy is worse than the disease causing enteritis and death.

#### 7 Poisons used to Simulate Injuries

*Plumbago rosea* Linnæus (Plumbaginaceæ) and *Anacardium occidentale* Linnæus (Anacardiaceæ) are used to simulate bruises and other injuries in order to get innocent people into trouble.

#### 8 Aphrodisiacs

The aphrodisiacs which are much in demand by tropical natives may be divided into those of animal and those of vegetal origin.





*Datura fastuosa* (Solanaceæ)—Poisoning by datura is common in India having been used by the Thugs. The seeds are usually mixed with food or drink and the symptoms which develop rapidly depend upon the dose being generally those of a cerebral poison. First there is delirium and later coma but in both conditions the pupils are widely dilated. The delirium is peculiar



FIG. 12.—*Datura fastuosa* LAMOUR.

for example the people affected may be found searching their bedding most vigorously for some lost article. When death occurs it is due to cardiac failure. The fatal dose is not actually known but may be about 10 to 15 grams of the seeds. Widdell puts the mortality at about 18½ per cent. The post mortem characteristics are wide dilatation of the pupils, congestion of the brain meninges and the lungs and other viscera.

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The symptoms are severe pain in the mouth and stomach, followed by collapse. The lips and tongue become swollen and blistered, the abdomen tender, the pupils widely dilated, while

may be given

*Fontainea puncheri*, Heckel, 1870 (Euphorbiaceæ), is a tree growing in New Caledonia, the ingestion of the fruit of which causes symptoms analogous to those produced by *Hippomane mancinella*.

*Illicium*—Guerreras and de la Paz have also drawn attention to poisoning from a decoction of *sanki*, which is the fruit *Illicium religiosum* v. Siebold, which belongs to the genus *Illicium* Linnæus, of the family *Magnoliaceæ* De Candolle, 1818. Montel in Indo-China has also found it to be poisonous. The symptoms appear to resemble cholera, but diagnosis has to be made from strychnine poisoning, tetanus and cerebro spinal meningitis. The symptoms were vomiting, diarrhoea, thirst, unconsciousness, convulsions, cramps, profuse sweating oliguria and anuria, small rapid pulse, cold extremities, paresis of the lower limbs, and exhaustion. The head is retracted, the eyeballs bulge, and the face becomes cyanotic when the respiration stops. In China and Japan it and its related species *I. anisatum* the star anise, which is harmless, are called badiane.

The seeds of *Ricinus communis* Linnæus (Euphorbiaceæ), the castor-oil plant, are poisonous, causing burning in the throat and abdomen, vomiting, purging (may be absent), and collapse. The fatal dose appears to be three seeds, and to kill in about forty-six hours. Post-mortem the principal feature is gastro intestinal inflammation.

The treatment is emetics, stimulants, and hypodermics of morphia.

### III. STIMULANT AND SEDATIVE POISONING.

Many drugs are employed all over the world to stimulate or to deaden the nervous system. These stimulants and sedatives have been used by man from time immemorable to whip up a flagging nervous system, or to deaden the effects of mental or bodily suffering.

Used judiciously and in a proper manner there can be no doubt that they alleviate human suffering, but if used injudiciously or immoderately, and especially if they are constantly taken, they become 'habit poisons,' and as such affect the cells of the body injuriously, and by so doing some of them become true 'racial poisons,' and as such have been mentioned in the section on Eugenics (see p. 118).

West Indies and South America will cause a painful swelling of the tongue if chewed.

The emanations from the flowers of *Arum dracunculus* cause dizziness headache and vomiting in some people.

*Blighia*—The succulent aril of the akee tree which belongs to the genus *Blighia* Koenig synonym *Cupania* Plumer (Sapindaceæ) and called *B. sapida* is used as an article of food in the West Indies.

It is however known that if eaten in an unsound condition it is poisonous and recently Scott has shown that this is the cause of the vomiting sickness of Jamaica as will be detailed in Chapter LVIII p 1695 of this book.

*Capsicum* (*C. annuum* Linnæus 1775 Solanaceæ) if taken in large quantities may cause burning in the mouth and throat vomiting colic diarrhœa and even death. *C. frutescens* Linnæus

to cause fatal effects by administration non maliciously of two dessertspoonfuls of its milk in a quantity of cows milk. This plant is variously named mudir in Bengal yercumbly by Tamils and warra by the Sinhalese.

*Chaetelia toxicaria* Don (Chaeteliaceæ native name magbari or manuch) the powdered fruit of which is used for killing rats has been described by Renner in Sierra Leone as the cause of poisoning in a Mendi carrier.

The symptoms were mainly vomiting diarrhœa trembling general weakness and inability to walk because the legs were paralyzed the tendon reflexes having disappeared. Hyperæsthesia was present over the inner side of the thighs and legs and pressure on the calf muscle gave severe pain. The bladder rectum and pupils were normal. The man recovered in about two months.

Renner remarks that this case is probably the clue to attacks of sudden paralysis of the lower limbs in young persons (twenty to forty years) of both sexes in Sierra Leone. When death ensues it is from paralysis of the muscles of respiration.

*Dioscorea*—Guerreras and de la Paz say that this genus as well as *Jatropha Anamista* *Stylnos* and *Datura* are the cause of poisoning in the Philippine Islands.

*Fungi*—The presence of poisonous as well as edible fungi should be remembered in the tropics although there is no literature known.

1 the matter forcibly

fragrance is said to have been their deaths for it looks like an apple not known

black probably due to some chemical change produced by the action of lime and saliva on cocaine. Pernicious symptoms in the form of emaciation accompanied by insomnia, digestive disturbance, diarrhoea, deafness, diminution of urine, delusions and hallucinations and even at times acute mania may appear.

*Indian hemp* (*Cannabis sativa*) is used in India, Arabia, Persia and elsewhere as a narcotic. It must be remembered that *C. sativa* grown in India has quite different properties from the same plant grown in Europe. In India there are four varieties sold: *bhāng* consisting of the dried leaves and stalks reduced to a powder; *ganja* the flowering tops; *charas* the resin from the leaves; and *majun* a sweetmeat prepared with hemp. In Central Asia the resin is called *haschisch*.

Its action on man is first to produce a pleasurable excitement and later narcotism. In moderate doses it temporarily increases the feeling of strength and power. As an intoxicant it is much used, being eaten or smoked, and is said to be one potent cause of insanity in India. Waddell says that about 38.4 per cent of Bengal lunacy can be traced to this source. It is asserted to be one of the causes of the strange phenomenon called *running amok*, but this will be dealt with in detail later on under diseases of the nervous system.

*Kava* is an intoxicating drink prepared from the root of *Piper methysticum* Forster (Piperaceæ) which grows throughout Polynesia.

According to Lewin *kava* contains starch, flour, two inactive substances—*kawun* and *yangonun*—and 2 per cent of resin which is believed to be the active principle. But this resin by treatment with ether can be resolved into two separate substances, one of which has a weak and the other a strong action on man. The drink causes a feeling of *bien-être* but too much has an evil effect with symptoms of inco-ordination and headache and a desire for sleep and may induce liver disease, dermatitis and general debility. Taken in moderation it is said to be harmless and also to be a cure for gonorrhoea and as such was introduced into Europe.

*Peyotl* (pellote) is a narcotic used in Mexico and derived from the cactus *Anhalonium lewinii* Henn. It causes sleep with hallucinations.

*Coriaria* species—*C. ruscifolia* Linnæus the poison foot of New Zealand and *C. myrtifolia* Linnæus (a native of Europe)—are considered to be intoxicant or poisonous according to the dose. The symptoms are coma, convulsions and dilated pupils. From *C. ruscifolia* the Maoris are said to prepare a wine and jelly.

*Pituri* (*Duboisia hopwoodi* F. Mueller—Solanaceæ) is a shrub growing in Australia and New Caledonia. In the former it is found in the neighbourhood of Carlo or Mungerebar on the Upper Mulligan and from thence in scattered patches eastwards. At Carlo live the Ulaolunga tribes from whom the other tribes purchase

It is not our intent to write except in the briefest manner upon these poisons as their effects are fully considered in special works and are also contained in all the ordinary textbooks dealing with general medicine. Notwithstanding this we feel that a few remarks dealing with these drugs in the tropics are necessary.

*Alcohol*—We have already referred to our belief that this is a personal and a racial poison and we would warn our readers not to be misled by statistics on this point and if they wish further information to refer to Adams on Karl Pearson (*and* references)

accident wards of the hospitals were nearly empty in the prescribed regions during the period of a social prohibition.

More dangerous in our opinion are the imported cheaper alcoholic drinks such as the cheap whiskies, gins and rums which being cheap are drunk in quantity and greatly deteriorate native races. These bad effects are not due to fusel oil which is not present in these cheap forms of spirit which in many ways are the purest form of alcohol to be obtained and hence their effect is truly due to alcohol and to alcohol only and their real danger is simply because they are cheap. *Methyl alcohol* is a direct poison to man a fact but little understood at the present time.

*Opium* is eaten in Persia, India and Africa and smoked in Malaya, Indo China and China but for the latter process it has to be specially prepared. On the Eastern mind opium is said to have two possible effects either it produces a sense of absolute blank or it produces fancy dreams and visions. The effects of chronic morphinism are loss of appetite, emaciation and exhaustion and hence inability to think or work.

It is however, probable that the effects of opium are not as bad as those of alcohol and used in moderation it may not be more harmful than the use of tobacco.

*Cocaine* has been used much of late in India as an intoxicant or stimulant to counteract the effects or in lieu of opium owing to the restrictions on the sale of opium. Unfortunately children have begun habitually to use the drug. The cocaine is chewed with betel and chunam (slaked lime) and produces at first loss of sensation in the tongue and lips followed by dryness of the mouth and fauces. The temperature does not rise but the pulse becomes full and quick and at this stage the inebriate likes to be left alone and firmly closes his lips lest the saliva should flow out. His ears become hot and red, his cheeks pale and the tip of his nose cold. Perspiration breaks out and the maximum amount of the so called hilarity or exaltation due to overstimulation of the nervous system now appears but is speedily followed by depression which induces the victim to take another dose. The teeth and tongue of the confirmed cocaine eater turn jet

because St Hilaire cancelled his original name. It is prepared by slightly scorching the leaves, which are then broken down and subjected to a strong pressure. A handful of this pressed foliage is infused in a small spouted vessel called a mate. It is then sucked hot through the spout or bombilla which is perforated on its lower side with small holes which while allowing the escape of the liquid prevent the pieces of leaf following. It is drunk freshly infused and is said to be an aperient and diuretic and to become a habit with those who drink it.

Mate is much used in Paraguay Uruguay the Argentine and Southern Brazil but in the last named *Ilex gongonha* Martius and *Ilex thecyrans* Martius are employed.

*Coffee*—The deleterious effects of excessive tea drinking are well known and need not be repeated but it may perhaps be as well to invite attention to the excessive amount of Turkish coffee which is drunk in the Middle East.

*Other Poisons*—In Malacca the leaves of *Mitragyna speciosa* Korth (Rubiaceæ) are said to be used in place of opium. From the leaves of *Bassia latifolia* Roxburgh and *B. longifolia* Linnaeus (Sapotaceæ) intoxicating drinks are made. *Hyoscyamus muticus* is used as an intoxicant by the Baluchians and makes them dance like lunatics.

The juice of the fruit of *Anacardium occidentale* (Anacardiaceæ) is used after distillation in Goa as a drink.

#### IV POISONS USED IN TRIAL BY ORDEAL

##### POISONS D'ÉPREUVE (FRENCH)

Curious customs exist in savage lands of trial by ordeal in which the patient is given a drug and then ordered to perform some act. Waddell records the history of an old Hindu woman who was supposed to be a witch. She was tried by ordeal being given a poisonous drug (datura) in treacle as it is a native belief that a witch can withstand poison. The result of the ordeal was that the poor woman died.

In Africa the greater part of these poisons belong to the Loganiaceæ Apocynaceæ Leguminosæ and Solanaceæ but the plant employed varies in the different regions and many are still undetermined—*eg* M Faug may be a strophanthus while M Boundou is undetermined.

Rho gives an account of trial in West Africa by *imbundi* the sliced root bark of *Strychnos scaja* (Baillon) which is said to contain strychnine. The accused after drinking a concoction of the root is made to jump over a stick and is pronounced guilty unless he is able to do this or to pass urine on to a banana leaf both of which feats are usually impossible.

Christison mentions the Calabar bean of West Africa *Physostigma venenosum* Balfour (Leguminosæ) as being used in trial by ordeal.

the pituri by barter with spears boomerangs etc About the beginning of March the pituri leaf is gathered and is sold in the form of half green half yellow tea mixed with plenty of chips. The preparation is complex it is first roasted in ashes then wetted with water then teased with the fingers and all larger pieces removed. Then leaves of a species of wattle or gidgeon are heated over a fire and finally burnt the ashes being retained and mixed with the moist pituri on a pituri plate — i.e. a smooth surface — and finally manipulated by the fingers into small rolls  $2\frac{1}{2}$  inches long by  $\frac{1}{8}$  inch thick. These rolls are chewed by the natives and are in great demand as a narcotic. Reserve rolls are carried at the top of the ear. The Australian native names are *Mijl* (Walookera) *ne-em pi* (Yaroungi) *un da kor a* (Undelere bina) *pi tu ri* (Ulaoinga) *ti rum bol i* (Karangi) *ti rem* (Mijl). The alkaloid piturine

is extremely common practice. *Wica belle* (piper betel) are *oberaca* (Pinang or areca nut palm) and mixed with lime. Betel stimulates the salivary glands and it is said those of the digestive organs. It diminishes the perspiration and should be spat out and should not be swallowed. The irritation may be the cause of the commonest cancer of all people in these parts. In the young it may possibly be the cause of heart and nerve diseases.

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*cellata* Thorne and *St sphaerocarpa* may also be noted. This nut has been known since the days of Leo Africanus in 1556 when it was known in the Sudan as *goro*.

It is supposed to be a nervous system and cardiac stimulant. It raises the blood pressure and increases muscular power and certainly with kola nut and water a Hausa can travel far and work hard as we know personally.

Analyses have been made and a glucoside *kolman* has been obtained by Hilger and Knebel.

*Guarana* — This substance which is made into oblong or round cakes is sold in Brazil as guarana bread being considered an indispensable requisite for travellers. It is made from the seeds of *Paullinia sorbilis* Martius (Sapindaceae) which are pounded and sweetened.

which Theodore consider guarana

*Male* — Paragu it is drunk or be

was the first to find and name the *lex paraguayensis* and this is accepted Lambert in 1824 changed to *paraguayensis* and this is accepted



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Africa The poison which is called waba wabajo or ouabaino was first fully described by Burton in 1856 It is prepared from

Gold Coast is probably derived from some species of *Acocanthera*. In Erythraea and Yemen *A. deflexa* Schweinfurth is used and in Somaliland *A. ouabaino* Cathelmeau. These trees are 4 to 5 metres in height with dark green foliage white or red flowers and violet red fruit. The poison is usually prepared by making a decoction of the wood and evaporating it down until it becomes a thick tar like extract which contains the active principle. This principle which is a glucoside called ouabain, acocantherin or wabain is a powerful cardiac poison. In addition the natives generally add snakes heads and gall bladders to the tar like mass but it is doubtful whether these really increase its virulence though it must be admitted that sometimes there are symptoms analogous to snake poisoning.

The thick extract of the wood containing any other additions which individual peculiarity may consider necessary is now painted upon the arrow heads.

The action of the freshly prepared poison is very rapid death taking place in a few minutes through stoppage of the heart after a preliminary quickening of the respirations and convulsions. Sometimes pain is complained of in the lumbar region. The symptoms can be readily prevented by a 3 per cent solution of permanganate of potassium. The native remedy is believed to consist in eating some of the poison.

Another important *Acocanthera* poison is *A. venenata* Thunberg which is employed by the South African Bushmen and is said to be made from a decoction of the bark. The symptoms are rigors without convulsions and loss of muscular power followed by death in a few minutes.

*Strophanthus Arrow Poison* Livingstone was probably the first to draw attention to a *Strophanthus* arrow poison called Kombi used in Central Africa. *Strophanthus hispidus* De Candolle is a very common plant in many parts of West and Central Africa and is a common arrow poison but not nearly so deadly as that of *Acocanthera*. The other varieties used are *S. glabris* *S. kambe* *S. lanosus* *S. ciabe* *S. barika*.

The poison is obtained by cooking the seeds in water and adding snakes heads and leaves and roots of other plants. The injured man falls to the ground and his breathing and pulse become gradually slower and slower until the heart beats suddenly cease and death ensues preceded by a convulsion in about ten to fifteen minutes. The heart stops in systole and will not contract on

the belief being that the innocent vomit and are safe while the guilty retain the poison and die. Its antidote is atropine administered hypodermically.

*Erythrophloeum puliciale* Procter *Faughimia reverifera* Poiret (which contains a toxic base with an action like digitalin) and *Menabea reneniti* Baillon are used in Madagascar and *Adeuun somalise* Poiret in Somaliland for purposes of trial by ordeal.

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The disease is acquired in two ways either by direct contact with the lacquer, or through the fumes arising from it by evaporation, but only as long as the lacquer is not dry, for the poison, whatever it is disappears on drying

The symptoms, which develop in a few hours are fever, with tension and oedema of the skin of the face, limbs and generative organs, nasal and conjunctival catarrh while a papular eruption appears on the oedematous skin of the legs and forearms

The treatment consists in washing the skin thoroughly with soap and water and applying soothing applications such as cold lotions, or *Lotio Plumbi subacetatis*

As prophylaxis the Chinese rub the hands and face with rapeseed oil in which a ham has been boiled and wear a linen mask for the face and a leather apron for the body After work the exposed parts are rubbed with a decoction of chestnut, pine bark, saltpetre and amaranth

The above precautions are taken in China, but in Japan no such prophylaxis exists

NOTE—Camel poisoning caused by the hydrocyanic acid contained in immature dairy is well known in the Sudan

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#### Trade P

CROCKER (1903)	<i>the Skin th</i>	
SCHREIBER (1903)	<i>Warm Climate</i>	

stimulation. *Strophanthus* is used on the Congo Lake Nyassa, Zambesi Gaboon Guinea Coast Gold Coast, Cameroons, and Senegambia as an arrow poison.

**Adonium Arrow Poison**—There are two species of *Adonium* used in Africa—viz, *A. boehmianum* Schum and *A. somalense* Oliver. *A. boehmianum* which is a shrub about 1½ to 2 metres in height is used by the Ovambo of German South West Africa to prepare an arrow poison called echuja. The thickest branches or roots are cut across and held over a fire, when the thick viscid sap exudes in threads and is collected by winding it round small pieces of wood. The arrows are then dipped into the sap and the sap dries upon them.

irritant cardiac

principle is an  
est slowing and  
om stoppage of

breathing. It is used by the Pigmyes of Central Africa but the principal Pigmy arrow poison is a mixture of this with strychnine which will kill an elephant. Prompt treatment however is said to be able to save a man's life when wounded by one of these arrows.

**Munchi Arrow Poison**—The Munchi arrow poison which is used by the Backorana clan in Northern Nyent is said to be nearly always fatal to man in about half an hour. The method of preparation is not known but the poison is plastered in a thick layer on the long point of the barb. It is brittle of a dark brown colour with slightly aromatic odour and is insoluble in cold or warm water in normal saline or acidulated solution but dissolves easily in alkalis—e.g., 1 per cent  $\text{Na}_2\text{CO}_3$  solution. It has no alkaloidal properties.

No  
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action on the muscle plasma can be detected. The toxic substance

be one of the in  
on) used by the

The other in  
gred ents are not known. According to Brieger the active principle is exactly the same as in *Acocanthera*. Rho says the Wakamba use a similar poison.

Less important African arrow poisons are—

(a) *Used by the Monballis Dwarfs*—

1. F—

siderable doubt about this matter as no scientific work has been done on the subject. The symptoms are gastro-intestinal irritation which may lead to collapse and death. Another fish which is only poisonous at certain seasons is a so-called sardine *Clupea*



FIG 14—*Clupea longiceps* (*Sardinella neohowii*)

*longiceps* (*Sardinella neohowii* Val) found off the coasts of Ceylon, and which according to Tennent caused much sickness years ago.

Other poisonous fish are *Tetrodon hispidus* the muki muki or death fish of Havan and the file fishes of which *Stephanolepis*



FIG 15—*Tetrodon vermicularis* SCHLEGEL  
(From Savitschenko's Atlas of Poisonous Fish)

*hispidus* L. may be mentioned as an example. Poisoning with *Tetrodon* has however been more accurately studied and may be referred to at greater length under the term Fuguismus.

### Fuguismus

The Japanese term 'fugu' includes several species of fish belonging to the genus *Tetrodon* of which the important species are *T. vermicularis*, *T. rubripes*, *T. laevigatus*, *T. chrysops*, *T. rivulatus*, *T. lunaris*, *T. pardalis*, *T. porphyreus*, *T. poicelonotus*, *T. stellatus* and *T. siconotus* which are said by Scheube to be often used for suicidal purposes. *T. pennantii* has also been found to be the cause of poisoning in a case in Burmah.

The poison appears to lie in the ovaries and testicles which according to Tabara contain—1. Tetrodin—a crystalline base of *Tetrodon* and a white, granular substance. Both are poisonous but

## CHAPTER XIII

### POISONOUS FOOD

Preliminary—Animal food poisoning—Products normally present—Iugurins  
—Post mortem decomposition—Vegetal food poisoning—Lathyrismus—  
Lolismus—Parapalmsis—Atriplicismus—Labismus—Lagopyrtismus—  
References

#### PRELIMINARY

Food poisoning technically called Bromatovismus may be divided into two classes—

- 1 Animal food poisoning
- 2 Vegetal food poisoning

#### I ANIMAL FOOD POISONING

Animal food poisoning is called zootrophotoxismus and may be due to—

- (a) Products normally present in certain animals but poisonous to man
- (b) Poisonous food having been eaten by an animal prior to its being killed for food
- (c) Products abnormally produced in the living animal
- (d) Post mortem decomposition

But of these we need only concern ourselves with the first and the last

**Products Normally Present**—Poisoning by products normally present in the animal is called siguatera and is generally due to fish though it may also be caused by Molluscs Crustacean and Coelenterates

The most common

are found among coral reefs and

It is possible that

such as the fish itself

or food such as dead

medusæ corals etc

Fritz and Branch have noted fish poisoning in 1915 in St Christopher and Nevis and think that it is due to the *Barraconda* (*Sphyræna*) while McNaughton reports similar poisoning from the Gilbert and Ellice Islands

Certain species of the genus *Clupea* (Cuvier) particularly *C. thrissa* (Osbeck) are noted as being very poisonous but there is con

*Dolichos filosa* called oordh dal is said by Kirke to be poisonous only if eaten with the husks when it causes colic indigestion and as secondary results rheumatic pains harshness and dryness of the skin with cracks. It is said to be a staple article of food among all classes except the highest and the lowest.

### Lathyrismus

**Synonyms**—Platterbsenkrankheit (Ger) Meurd Djuben (Algeria), Latirismo (It)

**Definition**—Lathyrismus is an intoxication caused by the ingestion of *Lathyrus sativus* Linnæus and other species of the same genus and c

**History**—  
the disease

who ate such bread suffered from a peculiar stiffness of their legs although they seldom died. In 1784 an epidemic was recorded in Tuscany when through scarcity of food the people were compelled to eat chick peas. Tozzetti while studying this epidemic came to the conclusion that only the people who had for at least three months eaten bread made of two parts chick pea to one part of rye or wheat became ill. He then planted some of the chick pea seeds and when they grew up identified them as *Lathyrus sativus* (L). In 1824 Desparanches came to the conclusion that the seat of the lesion was in the lumbar cord. In 1833 the disease was first recognized in India in the Sangor territories where on account of three successive famines in 1829-31 the people were compelled to eat vetches which are called kesari dal or teoni. Outbreaks took place in Sind Chota Nagpur the Central Provinces and in the Himalayas. It apparently became very prevalent for Irving says that in one district 6 per cent and in another 3.19 per cent of the inhabitants were affected. It still exists in India.

**Climatology**—The disease depends upon social rather than climatic conditions for people will not eat the vetches unless compelled by famine. It is known in India Algiers Italy and France.

**Ætiology**—There appears to be a consensus of opinion that the disease is due to eating bread composed largely of flour obtained from seeds of some species of *Lathyrus* belonging to the natural order Leguminosæ. The species most commonly suspected is *Lathyrus sativus* Linne which grows in India but *L. cicera* L (red vetch), *L. clymenum* L (Spanish vetch) *L. tuberosus* and *L. aphaca* have all been regarded as possible causes. It is however by no means evident what substance or substances in these plants cause the symptoms. Teilleux obtained a resin which caused tetanic spasms and paralysis of the posterior limbs in rabbits. Bourlier obtained an extract which killed birds and frogs. Asher obtained from *L. cicera* a volatile alkaloid which he called lathyrin which





**Diagnosis.**—Lathyrism must be distinguished from ergotism by the absence of gangrene from beri beri by the absence of implication of the peripheral nerves and the heart and the absence of dropsy

**Prognosis.**—The disease itself is not fatal

**Treatment.**—Mild cases may be considerably benefited by being given good food and warm clothing together with counter irritation to the spine and bromide of potassium internally in 15 grain doses three times a day Strychnine is harmful

**Prophylaxis.**—The only possible prophylaxis is the distribution of good food to the poor in times of famine

### Lolismus.

Lolismus is an intoxication caused by the ingestion of the seeds of *Lolium temulentum* Linnæus in bread

**History.**—Lolismus has been known since Roman times and is said by Orfila to have occurred in Genoa during the blockade of the year 1800 Kingsley of Roscrea described an outbreak in 1854 in which thirty persons suffered severely Similar cases have been reported in India from the Punjab where the herb is called mostaki and from the North West Provinces where it is called moschni

**Ætiology.**—The exact method by which *Lolium temulentum* causes disease is not known Dr Cordier experimented on himself by taking 6 drachms of the seeds early one morning and asserts that the result was inability to think indistinct vision torpor debility and drowsiness followed by efforts to vomit and later by tremors of the limbs great depression difficulty of speech and vomiting Bley separated a bitter principle which he called lolun but the action of this does not appear to have been investigated properly Freeman states that the seeds owe their poisonous properties to an associated symbiotic fungus which he carefully describes and says that it is probably identical with that found in other species of Lolium He says that it is a disputed point how far ergot and other fungi may be concerned in the production of the disease

**Climatology.**—It occurs in the Punjab and the North West Provinces of India and in France

ery giddy and  
me suffer from  
impairment of  
tation and in  
of burning heat

1  
symptoms and examining the bread when the starch granules of Lolium may be seen

**Prognosis.**—The disease does not end fatally

was doughy in consistence, alkaline, insoluble in water slightly soluble in ether, soluble in chloroform, and which on evaporation formed needles; but he did not perform any experiments with the substance the action of which is therefore unknown. It is believed by some that *Lathyrus* is not poisonous unless the seeds are decomposed or contain some parasitic growth, while others hold that the symptoms are not due to *Lathyrus* at all, but to *Agrostemma githago* (the corn cockle) or *Lolium temulentum* (the darnel). In 1883 Astier separated out an alkaloid *lathyrin* which Stockman in 1917 showed to be the poisonous principle, which is present in only small amount and only in the seed itself.

Animals are by no means immune from the baneful effects of the plants for ducks become paralyzed and may die after eating the seeds, while pigs and horses also suffer the latter showing acute or chronic symptoms which are said more or less to correspond to lathyrismus. On the other hand bullocks and buffaloes are con-

scarcity of food whether  
compel the unfortunate  
people to eat vetches instead of ordinary food. If the disease is brought about by famine it may assume epidemic proportions, if by poverty it may be simply endemic. Young people are more liable to be poisoned than old persons and men more than women perhaps because they eat more food.

**Morbid Anatomy.**—The pathology and morbid anatomy need

to a chill which he may have experienced a day or so previously.

Prodromata are often said to be absent though it is more probable that digestive disturbances colicky pains and diarrhoea do occur but pass unnoticed.

One of the first symptoms to arise is pain in the back and weak-

ness motion. There is no ataxy and no vasomotor phenomena, but the legs waste very much. The arms are not as a rule involved, though the hands may tremble. The superficial and deep reflexes are increased and ankle clonus is present. The electrical excitability of the affected muscles is diminished but the reaction of degeneration is absent. Incontinence of urine and impotence are early and common symptoms. The mind is unaffected.

The disease does not itself end fatally, but a definite improvement is seldom seen except in incipient cases.

Two varieties of *Paspalum scrobiculatum* are known in India—viz the sweet which is called 'pechadi' or 'goraharik' and is said to be wholesome and the bitter which is called 'dhome majaraharik' or 'mana lodra' and is considered to be poisonous. The poison appears to reside for the most part in the testa but the exact poisonous principle is not known. Barry points out that the seed is liable to the attacks of fungi and that perhaps the poisoning is really due to these parasites. It is clear that the whole subject requires revision.

**Symptomatology**—The symptoms are vertigo impairment of

I

which the flour is prepared

**Prognosis**—This appears to be on the whole good though fatal cases have been recorded

**Treatment**—Similar to lolism

**Prophylaxis**—Avoid the seeds of the grass in making flour

### Atriplexism

**Climatology**—The disease is as far as is known confined to China

**Ætiology**—In some way the disease is connected with *Atriplex serrata* (Chenopodiaceæ) which grows as a weed in the courts gardens and along the walls of the houses of Peking and is eaten by very poor people either uncooked or as a pancake

It is said never to be poisonous if well washed and if the red leaves are picked out. But Matignon drew attention to the fact that a small insect of a greenish yellow colour is found on the plant. It is possible that the disease may be due to this insect because Megnin found that *Holothyrus coccinella* Gervais a mite found in Mauritius and the Malay Archipelago causes severe inflammation of the part touched. This insect may be carried by the hand to the mouth. Laveran thinks that the people get the thumb and forefinger infected while plucking the herb and that it is by the hand that the disease is carried to the mouth. The ætiology is therefore extremely doubtful and it may be either an animal or a vegetal poisoning.

The predisposing cause is scarcity of food which compels the poorer classes to eat some of the best food available.

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unknown

**Treatment.**—Castor oil must be given to remove the poison and at the same time stimulants must be used to counteract the collapse.

**Prophylaxis.**—Bread should not be made with the seeds of *Juncus temulentum*.

### Paspalismus.

Paspalismus is an intoxication caused by eating bread made from flour derived in part from the seeds of *Paspalum scrobiculatum* Linnæus.



FIG 16 —*Paspalum scrobiculatum* LINNÆUS

Seeds show separately at the top of the illustration

**History**—Poisoning by *Paspalum scrobiculatum* occurs in India where it was reported as far back as 1879 and probably was known earlier.

**Ætiology**—Some authorities doubt the genuineness of this disease for as Waddell points out the symptoms are so like lolo mus that it is quite possible that some mistake may be made as to the causation.

cysts explode and the filament is ejected and pierces the skin of the animal attacked and so introduces the poison

**Zoanthari** *plongeurs*

Dr SKLIOS ZEROS the first symptoms are itching and intense burning in the place where contact with the anemone has taken place. A papule then appears surrounded by an area which at first is red but may become blue and finally black and may spread over the surrounding skin to a distance proportional to the virulence of the poisoning. The skin sloughs and leaves a suppurating ulcer. Dr ZEROS has produced the symptoms of this disease in a dog by rubbing an actinia held in forceps along its abdomen.

C RICHET has separated two poisonous principles from *Anemone scullatus*—viz thalassin and congestine. Thalassin is not very toxic producing cutaneous redness intense congestion of mucous

The application of fat to the skin is said to be a preventative to the venomous action of the anemone.

**Millepora**—Jones has described an acute erythema with severe pain followed by papules pustules and desquamation as the result of stings by the hydroids of the hydrocoralline millepores (*Millepora alcicornis*, *M. complanata* and *M. verrucosa*) in Malaya where the corals are known as karang gatal or itchy corals.

**Trachymedusæ**—The jelly fishes of European waters such as *Rhizostoma pulsum* of the Mediterranean and *R. cuvieri* of the English Channel are well known to cause local redness swelling and urticarial eruptions.

The jelly fishes of the tropics produce the same symptoms but with greater severity. The pain is agonizing and there is collapse

internally and to apply  
affected area. Usually

recovery is quick and there are no after effects.

Meyer describes a case of poisoning due to the well known *Physalia pelagica* (the Portuguese man of war) in which there was severe inflammation and fever. A similar case caused by *Cyanea capillata* has been recorded by Forbes.

Porter and Richet obtained a liquid from *Physalia* containing an active principle hypnotoxin which when injected into animals caused somnolence and finally death due to cessation of respiration.

**Symptomatology**—The disease begins suddenly without prodromata some ten to twenty hours after the plant has been eaten. The tips of the fingers and the back of the hands begin to itch though sometimes the irritation may be at first limited to the thumb and forefinger. The affected parts soon become painful swollen cyanosed and cold while the pruritus and swelling spread up the hands on to the forearms. The face and eyelids itch and in due course become swollen and the nose becomes cyanosed and cold. Sensibility to touch is diminished in the affected parts but much increased to heat and to the sun's rays. Blisters and ulcers may appear on the affected parts the latter often giving rise to keloid scars. The eruption may appear in any part of the body but is also occur

gnosed from  
should be no

immunity because in the former there is no oedema and in the latter there is redness as well as oedema.

**Treatment**—The treatment is symptomatic and consists in applying anodynes and cold compresses locally and giving purgatives and disinfectants such as salol internally. Good food and good hygiene are also requisite.

**Prophylaxis**.—The aetiology must be settled before definite rules for prophylaxis can be given.

### Fabismus.

**Synonyms**—Favism Il Favismo Bohnenkrankheit

**Definition**—Fabismus is a disease attributed to the eating of fresh beans (*Vicia faba*) or to the scent of the flowers thereof when in blossom.

**Historical**—It has been known for many years in Sardinia and has been carefully studied by Ferri in 1905 and then by Frongia in 1907 followed by Zoja in 1914 and Gasparri in 1915.

**Aetiology**—The actual cause is unknown but it is associated with the period of the year when the bean is ripening. It seems to appear only in certain families and there is a personal idiosyncrasy and no immunity is confirmed by an attack in susceptible persons who may have repeated seizures. It occurs in Sardinia and it is suggested that it may be found in other Mediterranean countries.

**Symptomatology**—Within a few hours of eating the beans or of being exposed to the scent of the flowers an acute febrile attack associated with marked blood destruction sets in. The red cells fall to 2 000 000 per cubic millimetre and the hæmoglobin to 20 per cent and icterus sets in with liver tenderness but no enlargement of that organ or of the spleen. Bile may be vomited and passed in the motions while hæmoglobin urobilin and indican are found in the urine. Children die in a few days but adults recover quickly.

cysts explode and the filament is ejected and pierces the skin of

ing in the place where contact with the anemone has taken place. A papule then appears surrounded by an area which at first is red but may become blue and finally black and may spread over the surrounding skin to a distance proportional to the virulence of the poisoning. The skin sloughs and leaves a suppurating ulcer. Dr Zervos has produced the symptoms of this disease in a dog by rubbing an actinia held in forceps along its abdomen.

C. Richet has separated two poisonous principles from *Anemone scintillans*—viz thalassin and congestine. Thalassin is not very toxic per membran lent than will kill a nistic to an other.

The application of fat to the skin is said to be a preventative to the venomous action of the anemone.

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internally and to apply affected area. Usually recovery is quick and there are no after effects.

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## ECHINODERMATA.

The Echinodermata possess poison glands which supply a venom to certain species but this only affects small animals.

The Cuvierian organs of certain Polynesian species of the holothurians

## PLATYHELMIA

*Dibothriocephalus latus* which causes a profound anemia is suspected to secrete some form of poison and indeed this theory is supported by certain experiments of Schramm and Tallquist. These investigators found that if the worms were subjected to tryptic digestion and then mixed with food and given to dogs by the mouth or extracted with normal salt solution and injected hypodermically, an exhaustion which ended in death was sometimes produced. In one case there was a great reduction of the

poison which can be obtained by pulverizing the tania with sand and extracting with normal saline solution. This extract was then filtered and injected into animals but the symptoms were not characteristic. Picou and Ramond consider that the extracts they obtained showed a decided bactericidal action.

On the other hand, the rupture of an echinococcus cyst is well known to produce symptoms of the poison is not known if the dose is small, leading to fatal collapse, if the dose is large. Injected into animals the liquid acts as a cardiac poison causing death by stoppage of the heart in diastole, together with various other symptoms such as a fall of the blood pressure and temperature.

## NEMATHELMINTHES

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minutes or a dose of 2 cc

Cattaneo obtained  
ascarides to live in  
Boycott however,  
ascarides

With regard to *Ancylostoma duodenale*, there has been much



discussion as to whether it produces a toxin or not. The experiments of Whipple and Preti seem to establish the presence of a hæmolytic principle in the alimentary canal of the worm and those

the host through lesions in the intestinal mucosæ caused by the bites of the worms

## ARTHROPODA

The Phylum Arthropoda includes a number of types which are characterized by their capability of stinging. The forms which we are about to describe occur in Class III Arachnida Class V Chilopoda and Class VI Hexapoda of the classification given in Chapter XXVIII

### CLASS III THE ARACHNIDA

As the definition and classification of this class is given in Chapter XXVIII we have only to consider the recognition of the three orders with which we are concerned here —

#### I Abdomen segmented—

Tail stout and armed at the end with a sting (Scorpionidea)

#### II Abdomen unsegmented—

(a) Abdomen connected with the cephalothorax by a short narrow stalk (Aranea)

(b) Abdomen fused with the cephalothorax (Acarina)

#### I Scorpionidea

Scorpions abound in the tropics where they grow to a large size and are much feared because of the poisonous properties of their sting. The method of striking is to bring the tail forward over the body of the scorpion so that the curved spine on the last segment (telson) of the tail penetrates into the skin and inflicts the wound. On either side of this curved barb is an opening through which the duct from a poison gland discharges the venom.

It is probable that the poison of different kinds of scorpions differ qualitatively and quantitatively but on this subject little is known. Certainly the sting of the small European scorpion

## ECHINODERMATA

## PIATYHELMIA

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characteristic. Picou and Ramond consider that the extracts they obtained showed a decided bactericidal action.

On the other hand the rupture of an echinococcus cyst is well known to produce symptoms of poisoning but the chemical nature of the poison is not known. The symptoms in man are urticaria if the dose is small, peritonitis and severe cardiac symptoms leading to fatal collapse if the dose is large. Injected into animals the liquid acts as a cardiac poison causing death by stoppage of the heart in diastole together with various other symptoms such as a fall of the blood pressure and temperature.

## NEMATHELMINTHES

minutes of a dose of 2 c c

Cattaneo obtained a substance toxic to guinea pigs by allowing ascarides to live in sterile broth. Cao, Jammes, Mandoul and Boycott however failed to obtain any evidence of the toxicity of ascarides.

With regard to *Ancylostoma duodenale* there has been much

which are used  
which are used  
walking legs.

Scorpions seize their prey with the pincers, hold them close to

body, and allowing it to remain there until the poison has had time to act. The telson consists of two portions—a broad swollen part (the ampulla) and a narrow portion (the spine), near the extremity of which are two small openings for the escape of the venom.

The two poison glands lie inside the ampulla, one on each side of the middle line. Each gland is covered with a sheet of muscle on its mesial and dorsal aspect. This muscle which is called by Wilson 'the compressor,' is inserted by its edge mesially along the ventral inner surface of the wall of the telson, and by a broader insertion laterally. The compressor muscle squeezes the poison out of the gland, along the duct, and through the opening in the spine into the victim. The epithelium of the gland shows three distinct types of cells—the mucous cell, the fine oxyphile granular cell, and the cell with very large granules.

**The Venom.**—Scorpion-venom is a clear, faintly acid fluid of a somewhat

thick or oily consistence, and possessed of an extremely faint yellowish colour. It contains no structural elements, but crystals form in it if evaporation takes place. On an average it contains about 28 per cent of solids.

Wilson gives the following figures for the venom of *Buthus quinquestratus*.—

Specific gravity	1092
Solids .. .. .	20.3 per cent
Ash .. .. .	8.4 ..

Proteids form part of the solids, and it is believed that the active principle is either a nucleo-proteid, acid albumin, or a primary proteose. The effects described by various



FIG 17—*Heterometrus indus* (Beer)  
(A scorpion commonly found in Ceylon)

kill not merely children but even adults. According to Cr  
as many as 200 persons die annually from scorpion sting in th  
I a t r s

though the ancients were well acquainted with the sting its  
effects and had woven wonderful legends as to the nature of the sc  
animals. Redi experimented upon a pigeon and a dog. The real  
study of the venom began with Guyon 1844 Paul Bert 1855  
Delange 1866 and Valentini in 1871 and was followed up by  
Joyeux Laffine in 1883 and many others. A full literature is  
given in Faust's Die Tierischen Gifte.

Classification—This is unsettled but Pockl gives the fol  
lowing—

- I Pentagonal cephalothoracic sternum—
  - (a) Single pedal spur (Pantiniidæ Thorell 1856)
  - (b) Two pedal spurs (Vejovidæ Thorell 1856)
- II Triangular cephalothoracic sternum—  
With two pedal spurs of which the ante- or is bifur-  
cated (Buthidæ Simon 1879)
- III Short wide antero-posteriorly compressed cephalothoracic  
sternum—  
Two pedal spurs (Bothriuridæ Simon 1878)

Geographical.—Scorpions occur all over the world but the largest  
and most dreaded are found in the tropics. They live under stones  
under the bark of trees in sand and also in houses which they  
leave at dusk. Some of the best known are—

- 1 *Euscorpis carpathicus* Linnæus Vejovide (3 to 35 centi-  
metres long)
- 2 *Buthus afer* Linnæus 1764 (16 centimetres long) Africa Asia
- 3 *Buthus quinquestratus* Hemprich and Ehrenberg 1828 Upper  
Egypt and the Sudan
- 4 *Scorpio maurus* Linnæus 1758 Egypt Tunis  
and Alexandria (9 centi-  
metres long)
- 5 *Heterometrus inlus* Geer 1778 Ceylon

Anatomical.—The body of the scorpion is divided into a cephalo-  
thorax or prosoma behind which comes an abdomen subdivisible  
into a broader portion or mesosoma and a narrower metasoma  
with five segments which is popularly called the tail. At the  
end of this metasoma there is a postanal curved spine called the  
telson inside which lies the paired poison gland. The appendages

method by which it kills its prey which usually consists of small animals and further that in order to be toxic the venom must be injected subcutaneously or intravenously for by the mouth it is harmless.

When experiments are performed on animals the following symptoms appear —

- 1 Local irritation and pain
- 2 Muscular twitchings chiefly of the head and neck
- 3 Jumping movements
- 4 Lachrymation
- 5 Increased orbital nasal and salivary secretions
- 6 Muscular spasms especially of the hind limbs but also in all muscles
- 7 Erection of the hairs
- 8 Passage of liquid feces (often absent)
- 9 Erection of penis and emission of semen

The venom of *Scorpio maurus* causes death in small birds within two minutes to half an hour from failure of the respiration. If the venom of a scorpion is placed on the conjunctiva of a rabbit violent ophthalmia results.

In man the symptoms depend upon the size and nature of the scorpion. Thus the sting of the small ( $3\frac{1}{2}$  centimetres) *Euscorpheus europæus* causes only pain redness and local swelling whereas the larger tropical scorpions cause very intense pain of a burning character radiating from the skin associated often with violent convulsions mental disturbance and hallucinations profuse perspiration and secretion of saliva and perhaps vomiting. The pulse is weak and quick and the respirations hurried and shallow. These symptoms gradually diminish in three to eight hours and by about nineteen to twenty four hours the person is usually normal. This however is not always so for death may ensue due to collapse or stoppage of the respiration effects which are more likely to happen in small children than in adults. Thus Wilson states that the mortality in children under five is 60 per cent for *Buthus quinquestriatus* but the mortality diminishes as the age increases. Of course this is simply due to the greater dilution of the poison in the body of the adult.

In addition to the above symptoms some authors have described trismus but it is probably due to infection with the bacillus of tetanus. The erection of the penis noted in experiments on animals has been seen in man by Delange and Guyon in Algeria. A paralysis of the lingual and hypoglossal nerves has been noted by Posada Arango. Lymphangitis and adenitis are described as part of the local effect of the sting.

The above symptoms would indicate the action of a neurotoxin acting upon the nervous system and causing first of all increased paralysis of the medullary

are correct there are no

indicate the presence of toxins one resembling the neurotoxin of snake venom and another hæmolysin for Kyes has described a typical lecithide producing hæmolysis like the lecithides of cobra venom Calmette has also shown that the venom of *Buthus occitaneus* is neutralized by cobra antivenene. There would therefore appear to be some resemblance between scorpion venom (or at all events the venom of *Buthus occitaneus*) and cobra venom. It is however impossible to make any definite statements as the condition of our knowledge with regard to this poison is most unsatisfactory.

Ivano says that the poison is a protein of which there are two kinds one soluble in water and the other in dilute acids and from these crystalline bodies can be prepared. Lecithin and cholesterol are also present in the venoms which can be destroyed by pepsin and trypsin permanganate of potash and calcium hypochlorite. It seems to be very like snake venom and it is time that researches on modern lines were made.

Joyeux Laffine thought that the venom first increased reflex action and then caused paralysis of the nervous system and that death was due to a curari like poisoning of the end plates of the respiratory muscles but Valentin found these were quite intact and that the muscles contracted well when their nerves were stimulated by electrical or mechanical stimuli.

As regards the action on the blood coagulation hæmorrhage due to change in the capillary walls and hæmolysis have been observed as well as agglutination of the red corpuscles which are said to form viscous masses and thus to block the bloodvessels by embolism.

These observations were made by Jousset de Bellesme on *Lilla viridis* a frog remarkable for its lack of pigment and therefore specially suitable for such a purpose. If confirmed they would show the presence of fibrin ferment hæmorrhagins hæmolysins and agelutinins in scorpion venom and would make it resemble very closely snake venoms. Sanarelli however was not able to observe any change in the red cells beyond hæmolysis which he saw in the blood of fishes amphibia and birds.

In conclusion we may therefore assume the presence of a neurotoxin acting on the central nervous system and the presence of hæmolysins until further experiments give us more exact information.

**Minimum Lethal Dose**—The minimum lethal doses for dry

by the fact that the minimum lethal dose for a guinea pig is 0.1 milligramme per kilogramme which gives a toxic value of 10,000,000 for *Buthus quinquestratus* but as may be imagined the toxicity of different venoms varies considerably and the difference may be not merely quantitative but qualitative.

**Effects of the Venom**—It must be remembered that the venom is not merely a means of defence for the scorpion but it is also the

He considers that the secretion of the poison gland only gives rise to local symptoms and that the general symptoms are due to this toxalbumin and that it is because of this admixture in *Latrodectus* that the bite may cause severe symptoms and even death in human beings. The common European garden spider (*Epeira diadema*) only causes local irritation because the toxalbumin though present in the body is not mixed with the poison of the poison gland. He also describes a hæmolytic action in both *Epeira* and *Latrodectus*.

Sachs has contributed a paper in which he carefully studies this hæmolysis which he calls arachnolysin and Wilson has recently written an excellent monograph on the spider bites.

**Classification**—Spiders are divided into two suborders as follows—

- I Spinning organs set to far anterior to the anus. Eleven Mesothelæ)
- II of the anus. No

The Opisthothelæ are the only forms which concern us and they are divided into tribes as follows—

- A Only anterior pair of spinning organs present (Megalomorphæ)
- B Two pairs of venomous spinning organs present (Arachnomorphæ)

The Megalomorphæ include—

- I Without large maxillary process on the base of the palp—
  - 1 Feet furnished with apical tufts or pads of hair (Aviculariæ)
  - 2 Feet not so furnished (Ctenizidæ)
- II With large maxillary process on the base of the palp (Atypidæ)

The bird eating spiders (Mygale) come under the Aviculariæ

The Arachnomorphæ include—

- The Lpeiridæ with *Epeira diadema* the Therididæ with *Latrodectus* the Lycosidæ with the Tarantula spider

to be poisonous but the Chile *L. sceltio* the *ularia* L. South Ar *Chiracanthum* nutrix Walck. *Theridium tredecim guttatum* France and Italy

**Effect on the Scorpion.**—At the present time it is not believed that a scorpion commits suicide when in difficulties by stinging itself in the head with its own stinger. It is said to be known again

immunity by its own sting is said to be known

**Immunity.**—A natural immunity exists in the jerboa (*Faculus*

ja) but this has not so far been obtained in any animal. Calmette has reported that the cobra which is found in

immunity but this has not so far been obtained in any animal. Calmette has reported that the cobra which is found in

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#### 496. de JULIOWS —

Under one year	5
One to five years	8
Five to fifteen years	7
Total	<hr/> 20

Deaths of adults however are known but generally take place in a few days not quickly

**Treatment.**—In the treatment the first thing to do is to give a full dose of the serum, and then to apply a proximal ligature and to treat the wound with permanganate of potash as described under the

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solution of cocaine be injected subcutaneously close to the sting in an adult and 1 to 5 mmms in infants and children. Eucaine or stovaine might be preferable and can be imported from any chemist in sterile capsules ready for hypodermic injection. Simpson recommends the local application of a paste of ipecacuanha

## 2 Aranea

The Aranea or spiders are found all over the world but by far the largest are in the tropics and their peculiarly repulsive appearance has given rise to numerous fables both ancient and modern with regard to their poisonous properties



whether the cramps and convulsions are really due to action of the poison upon the nerve cells or merely to the altered blood conditions has not been decided

The reader is particularly asked to compare these actions on the nervous system (neurotoxin?) on the mucosa of the stomach on the capillary wall on the blood and red cells with the venoms of the scorpion and of the snakes which they strongly resemble

Acquired immunity can be produced in animals by injections of non lethal quantities of venom

**Minimum Lethal Dose**—The minimum lethal dose for cats is 0.20 to 0.35 milligramme of the dry venom per kilogramme of the body weight. Dogs are less sensitive and hedgehogs still less while frogs require fifty times the quantity of poison which will affect warm blooded animals

The symptoms of the bites of the different spiders will now be briefly described

**Bite of *Latrodectus mactans***—The symptoms of this bite are local pain which does not appear till some little time after the bite but becomes agonizing and may last for a couple of days. In addition tetanoid symptoms may set in but usually end in recovery in about ten days

**Bite of *Latrodectus scelo***—This is the katipo spider of New Zealand. The symptoms begin in about thirty minutes with the formation of a white vesicle surrounded by a red halo and severe pain at the site of the bite. The general symptoms include first stiffness of the muscles about the mouth and jaw so that it is difficult to open the mouth or to speak and impossible to swallow. The pulse becomes very slow (12 to 14 to the minute) and there is extreme pallor of the face and body with coldness of the extremities which are quite flaccid. Respiration becomes slower and slower and death may take place at this stage or an illness lasting about six weeks and somewhat resembling typhoid may ensue which may end either in death or recovery

The bite is characterized by smart

*T. lugubre* Koch Kara Kist of Russia *Segestria perfida* St *Chato*  
*pelina olivacea* *Lycosa tarantula* L. *L. singoriensis* Laxman  
*Epeira diadema* Walck.

**Anatomical.**—The body of the spider is sharply divided into cephalothorax and abdomen. The pairs of appendages are six in number—(1) The two-jointed chelicere (2) the six jointed leg like pedipalpi (3) the seven jointed legs. The poison gland usually lies in the basal joint of the chelicera ensheathed in connective tissue inside which there are two spirally arranged layers of non striped muscle surrounding a basement membrane which bears two to three layers of polyhedral cells surrounding the lumen of the acinus. From the gland the duct runs forwards into the distal hook shaped joint upon the apex of which it opens.

**The Venom.**—The venom which is useful to the spider enabling it to kill the small animals upon which it lives is an oily translucent lemon yellow coloured liquid with an acid reaction and a hot bitter taste. It has been reported to act on one and all of the ventila-

harmless by heating to 90° C and the active principles are said not to dialyze. The chemical peculiarities and the active principles of the venom are little known. Robert as has already been pointed out considers that there are two poisons—(1) A toxin secreted by the poison gland and only causing local symptoms (2) a toxalbumin distributed through the body (not originating from the poison gland) and causing general symptoms. The first exists alone in *Lycosa tarantula* *L. singoriensis*. The second largely predominates in *Latrodectus*.

Robert and Sachs have found and studied a hæmolysin arachnolysin in the venom of several kinds of spiders and Sachs has been able to immunize a guinea pig against this toxin and produce an active serum. Arachnolysin acts upon the red cells of man rabbit or mouse and goose but not on those of the horse dog sheep and guinea pig.

Spider venom is also said to increase the coagulability of the blood. The venom of *Theridium lugubre* is believed to act injuriously on the isolated frog's heart even when diluted to 1 in 100,000 but it is not known whether this is due to action directly upon the heart muscle or upon the local nervous apparatus. The walls of the capillaries are also said to be damaged by spider venom and to allow an increased amount of transudation and hence the hæmorrhages and œdema seen about the wound. It is asserted that the venom acts deleteriously upon the mucous membrane of the stomach and intestines causing redness and swelling and even hæmorrhages which perhaps are due to some attempt at excretion of the poison by these organs. It is also thought that the venom acts upon the central nervous system but

of secretion into the victim but the nature of this fluid and its action requires further investigation Nuttall drawing attention to the immunity following bites says that it is probable that this secretion is toxic in its action

**The Effects of the Venom**—With regard to the Argasidae *Argas persicus* Oken has an evil reputation in Persia where its bite is said to cause severe pain fever lassitude delirium convulsions and even at times death in new comers while natives are immune

Bordier considers that these symptoms are due to the injection of a poison but this would hardly appear to be likely as Lounsbury found in his own case in South Africa that the bite

caused only slight itching If the symptoms are properly described in Persia it would indicate that the tick introduced some parasite into the new comer which caused a definite disease to which the native had acquired an immunity

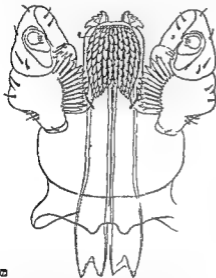


FIG 18—VENTRAL ASPECT OF THE MOUTH PARTS OF A TICK (*Hamamphysalis punctata* CANESTRINI AND FANZAGO)  
(After Nuttall Cooper and Robinson *Journal of Parasitology*)

1 Chelicerae showing teeth 2 hypostome showing rows of recurved teeth 3 palp



FIG 19—CHLICERA FROM THE SAME TICK

(After Nuttall Cooper and Robinson *Journal of Parasitology*)

1 Internal digit 2 external digit

*Argas reflexus* Fabricius may cause local pain and swelling with sometimes an erythematous eruption while the site of the bite is marked for years by a cicatrix The bite of *A. bruntpi* Neumann is also severe

*Ornithodoros moubata* Murray inflicts a very painful bite with much swelling and the formation of raised hard wheals in Europeans which may last several days

*Ornithodoros turicata* Duges may cause swelling and numbness all over the body with vomiting and diarrhoea accompanied by an urticarial eruption and profuse perspiration with rigors fever

commoner heading of *Mygale* and cause prolonged inflammation and extensive cicatrization. *Theraphosa javanensis* is reported to kill men.

*Bite of Chelopelma olivacea*—The local symptoms are great pain, redness, swelling, but whether a general effect (curari-like poisoning of the voluntary muscles and death from stoppage of respiration) takes place is very doubtful.

*Bite of Lycosa tarantula*—The bite of this spider produces wheal surrounded by a red areola, but no general symptoms result and tarantismus only exists in popular imagination. The tarantula dance was probably introduced as a cure, with the purpose of keeping the patient on the move so that he should perspire and thus get rid of the poison. The tradition of the Middle Ages was that the bite caused the dance frenzy, *Chorea saltatoria* or tarantismus, which was supposed to lead to such violent exertion that death resulted unless the victim was soothed by music.

The Bite of Tarantula—The bite of the tarantula spider has

burned in a charcoal fire, are dropped into a basin of cold water.

**Diagnosis.**—Bee-stings, scorpion bites, and ordinary skin bacterial infections must of course be distinguished from spider bites, with which, without doubt, they have been often confounded. The following may be some guide:

The bite of a spider is usually accompanied by a severe pain, which is proximal in nature, open to saline solutions—e.g., weak potassium permanganate, potassium or equal parts of spirit of ammonia and water as a wet dressing. It appears to us that a strong permanganate of potash solution ought to be given a trial.

### 3. Acarina

The Ixodoidea, or ticks, are well known to cause severe symptoms by their bites, apart from the introduction of any parasite such as a *Troposphora* or *Spirochaeta*.

The anatomy of these arthropods is given in detail in Chapter XXVIII, p. 689, to which reference should be made, but a few remarks are necessary concerning the act of biting, which has been studied by Nuttall.

The tick pierces the skin by means of the teeth on the digits of its chelicerae. The digit is capable of being extended by an internal muscle and turned outward by an external muscle. These movements, occurring alternately, cause the teeth to cut the skin and as the chelicerae work deeper and deeper, the hypostome is dragged into the wound, and by its recurved teeth keeps the tick in position. The palps but rarely enter the wound. During this act of biting, it is believed that the salivary glands pour a considerable amount

places especially in woods. The poison apparatus of the centipedes is formed by the appendages of the first trunk segment being modified so as to form a large pair of jaws at the base of which the poison gland lies. The duct of this gland opens on the apex of the claw and therefore as there are two jaws a centipede-bite will show two minute punctures or drops of blood.

The venom is primarily intended to kill their prey which consists of small insects and larvæ.

**Geographical**—France Spain Ital  
Africa India Indo  
Leach Mid Europe

**The Venom**—The venom is an acid opalescent liquid but little miscible with water. For experimental purposes it can be obtained by treating the lower lip and the hooks with normal saline solution.

When injected into the veins of rabbits it causes an immediate paralysis with coagulation of the blood while under the skin it forms a large abscess.

**Effects of the Venom**—The poison causes local and general symptoms. At first there is itching but this is quickly followed by intense pain which extends all over the limb. A red spot appears at the site of the bite which enlarges and becomes black in the centre and sometimes there are lymphangitis and lymph

and the

**Prognosis**—The prognosis is good though small children have been known to die from the effects of a sting. Adults as a rule recover in about twenty four hours at the most.

**Treatment**—Bathe the part well with a solution of ammonia (1 in 5 or 1 in 10). After bathing apply a dressing of the same alkali or if there is much swelling and redness an ice bag.

If necessary give hypodermic injections of morphia to relieve the pain. At a later period fomentations may be required to reduce the local inflammation.

### 3 HEXAPODA

The Hexopoda or insects contain many species injurious to man. The orders to which the principal venomous species belong are (1) Anopleura (2) Hemiptera (3) Hymenoptera (4) Lepidoptera (5) Diptera and (6) Coleoptera.

#### 1 Anopleura

This order includes the lice which cause much irritation by their bites. The nature of the venom however is not known and the lice are of more importance as carriers of disease and will therefore be dealt with more fully in Chapters XXX (p 749) and XXXV (p 872).

headache and backache if the poisons enter a vein. Locally an ulcer may form at the site of the bite. *O. talaje* Guerin Meneville causes severe itching and pain.

With regard to the Ixodidae *Ixodes ricinus* causes severe dermatitis which may be followed by pustules and abscesses with oedema lymphangitis and lymphadenitis associated with fever.

**Treatment.**—The treatment of tick bites is first to detach the tick which is by no means easy as the recurved teeth of the hypostome hold on to the wound very firmly. The best plan is

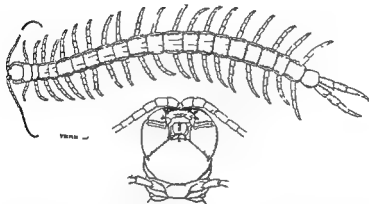


FIG. 6.—*Scolopendra morsitans* Linnaeus

As regards prophylaxis badly infected native huts should be burnt while ordinary houses may be fumigated with sulphur or carbon bisulphide or sprayed with kerosene or boiling water. Beds must always be raised from the ground and the feet of the bedstead placed in water containing kerosene while pyrethrum powder may be dusted between the coverings of the bed. Wellman insists that natives should not be allowed to sleep in or near the quarters of Europeans.

#### CHILOPODA

The class Chilopoda includes the Scolopendridæ or centipedes which are animals with a head and a uniformly segmented trunk possessing numerous legs. They are very common all over the world but the tropical species are much larger than those which inhabit the Temperate Zone. They live under stones in shady

During this process respiration is going on quietly, but at times, according to Schaudinn, a violent contraction of the abdomen is noticed, which is said to be due to the carbonic acid in the superficial layers of the skin for he found that when carbon dioxide gas acted upon mosquitoes a like contraction was produced



FIG 21 — AN ANOPHELINE MOSQUITO (*A maculipennis* MEIGEN) IN THE ACT OF BITING  
(After Nuttall and Shipley *Journal of Hygiene*)

As this process is proceeding an irritating substance the chemical nature of which is not known is injected under the skin, and it is evident that this happens before the mosquito begins to suck blood for the irritation is present when the

insect ceases to bite before drawing up the blood

There has been much dispute as to where this substance comes from, but this appears to have been settled by Schaudinn, who

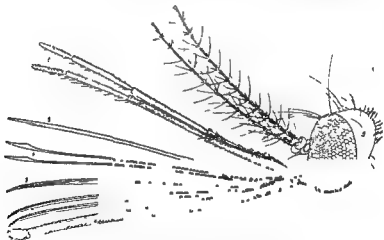


FIG 22 — HEAD OF *Anopheles maculipennis* MEIGEN  
(After Nuttall and Shipley, *Journal of Hygiene*)

1 Labrum, 2 maxillæ, 3 hypopharynx 4 mandibles 5 labrum,  
6 palpi, 7 antennæ, 8 occiput 9 eye 10 clypeus

trituated the isolated salivary glands in salt solution, which he applied to a wound with negative result On the other hand, when

2. Hemiptera

The Hemiptera (Chapter XXXI p 761) include the families of the Cimicoidæ or bugs and the Reduviidæ or cone noses. In the latter family is classified *Phonergates bicoloripes* Stal which according to Wellman produces a very painful bite in man in Angola.

It is said to be closely related to *Phonergates bicoloripes*.

Bugs—These hemipterous secrete a strongly alkaline poisonous secretion in their salivary glands. This poison flows down the ducts of the stylets and passing into the wound made by the bite dilates the capillaries causing an increase of blood in the area.

A more systematic description to be given especially as the nature of the venom is quite unknown.

3 Hymenoptera

In this order come the bees wasps and ants.

APIDÆ

The members of this family which sting are well known and it is by no means infrequent to hear of animals and even at times

wood bee

Historical—The venom of the bee was first studied by Brandt and Ratzeburg in 1833 then by Paul Bert in 1865 and Carlet in 1884 but the chemical nature was first investigated carefully by Jo of Langer in 1897 and in 1904 Phisalix made experiments on sparrows.

Anatomical—The body of the bee is divided into head thorax and abdomen from the posterior end of the last of which projects the sting in the form of a chitinous sheath narrow posteriorly and wider anteriorly. This sheath contains two barbed darts and into its wider portion (which possesses a cleft by which air can penetrate into it) two or three ducts from glands open. The principal opening belongs to the duct of the acid gland and opens anteriorly into a sac—the poison reservoir—which leads into a long slender coiled tubular gland either bifid anteriorly or subdivided into two glands. This long gland ramifies amongst the



**Symptoms.**—Shortly after a mosquito has bitten a person, a sensation of itching is experienced in the affected part, which on examination is seen to be inflamed and reddish, while a wheal not unusually develops, especially in persons new to the tropics. Sometimes a papule or even a nodule may form on the site of the bite, while more rarely scratching leads to secondary infection and the formation of boils, lymphangitis, or lymphadenitis.

**Treatment.**—The itching may be relieved by dilute solutions of ammonia (Scrubb's Ammonia is a favourite remedy) or by a 5 per cent solution of carbolic acid, or 1 per cent alcoholic lotion of menthol. Inflamed bites may be cleaned with 1 in 40 carbolic lotion and afterwards dressed with boracic ointment. Local septic poisoning should be treated by boracic or carbolic fomentations,

bites will be discussed

as the Culicidæ cause irritation by their bites, such as fleas, but the nature of the venom not being well known they will be considered together in Chapters XXXII, p 771, XXXIII, p 814 and XXXIV, p 857.

In Cape Colony there is a superstition that the 'bee moth'—i.e., death's head moth, *Acherontia atropos* Linnæus—is poisonous. This is not so.

## 6. Coleoptera.

Beetles and their larvæ are capable of inflicting severe bites or wounds by means of stiff hairs. *Silvanus surinamensis* L., the saw-toothed grain beetle, is said to bite people. Wellman describes the larva of a beetle which the natives of Angola call 'ochusia,' which means to be left alone, whose bristles will even penetrate the skin of the sole of the foot causing pain, inflammation, and even sloughing, when trodden upon.

**BLISTER BEETLES**—Chalmers and King in 1917 have drawn attention to the beetles *Epicauta sapphirina* MacKlin, 1845, and *Epicautat omentosa* MacKlin, 1845, as the cause of 'seasonal vesicular dermatitis' in Khartoum, while P. H. Ross had studied in

Dermatozoonoses, and is only mentioned here for the purposes of reference.

It will be noted that the various authors only mention two genera—viz., *Epicauta* Dejean, 1803, which belongs to the family *Cantharidæ* Leach, 1817, and *Pæderus* Fabricius, 1775, belonging to the family *Staphylinidæ*, which latter lack the appendage

he applied the isolated cesophageal diverticula to a scratch he obtained the characteristic irritation and redness. These cesophageal diverticula contain gas bubbles and bacteria or moulds. The bubbles were shown by Schaudinn to contain carbon dioxide by applying baryta water to the diverticula when a precipitate was obtained. The fungi need further investigation but they or their products appear to be the real cause of the irritation for



FIG 23—ANTERIOR END OF A MANDIBLE

(After Nuttall and Shipley *Journal of Hygiene*)



FIG 24—ANTERIOR END OF A MAXILLA

(After Nuttall and Shipley *Journal of Hygiene*)

when Schaudinn pressed the carbon dioxide out of the sac the signs characteristic of the bite were still produced. It appears probable therefore that the powerful abdominal contraction mentioned above expels the gas, bacteria and fungi from the cesophageal

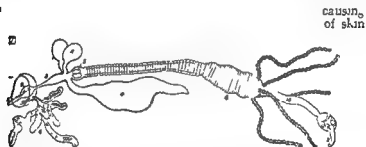


FIG 25—THE ALIMENTARY CANAL OF *Anopheles maculipes* MEIGEN

(After Nuttall and Shipley *Journal of Hygiene*)

1 Mouth and buccal cavity 2 pharynx 3 cesophagus 4 dorsal and ventral cesophageal diverticula 5 proventriculus 6 salivary glands 7 narrow portion of ventriculus 8 so-called stomach 9 malpighian tubules 10 intestine 11 rectum 12 anus

affected and thus enable the mosquito to get her supply quickly for feeding only takes two to three minutes during which time some mosquitoes will so overfill themselves with blood that it may be ejected *per anum*. The use of the carbonic acid is probably to prevent the coagulation of the blood which is drawn up the large blood tube formed by the labrum epipharynx and hypopharynx by the suction action of the pump like pharynx.



COLEOPTERA

on the ventral aspect of the claws which is s  
former

The life history of these beetles is very co  
of it is spent under the ground hence the  
eruption caused by cantharidin which is  
exuding from the insect and especially from  
the knee joint when irritated

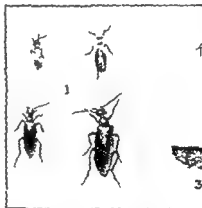


FIG. 6. BISTER BEETLE

1 Upper and smaller *Epica laetitia* Maelk  
larger *Epica sapphirina* Maelk 1845 2 *Vyl*  
1857 3 a claw of *L. sapphirina* to show the long ap  
closely applied to it

MOLISCA

A venomous snail of unknown genus in f  
in the Solomon Islands. These snail has  
with pointed tubular teeth armed with  
teeth are connected with a poison gland and a  
men and producing severe wound. Bite f  
(Terebridae) are said to be poisonous

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## CHAPTER XV

# VENOMOUS ANIMALS (*continued*)—PISCES AND AMPHIBIA

Pisces—Zoological classification—Geographical distribution—Poison by bite—Poison by stings—Amphibia—References

### PISCES

ALL over the world but especially in tropical seas there are fish which for purposes of defence secrete poisonous fluids from special glands. These fish have been but little studied and still less is known about the nature of their venom and its physiological action. A great deal however is known by residents in the tropics about wounds inflicted by these fish and the doctor practising therein is bound sooner or later to come across not merely persons who know a good deal in a general way about the subject but those who either are suffering or have suffered from the poisoning. It is therefore necessary that the tropical practitioner should have some

based on the manner in which the fish inflicts the poison  
Venomous fish may be classified into —

- Class I Fish which poison by their bite
- Class II Fish which poison by barbs (spines) connected with special glands
- Class III Fish which poison by a secretion prepared by the skin glands. This class is illustrated by the lamprey which is only known to produce poisoning when eaten

It will be understood from the above classification that venomous fish are to be distinguished from poisonous fish—i.e. from fish which cause symptoms of poisoning when their flesh is eaten—for the flesh of venomous fish can be eaten with impunity. The poisonous fish and their effects have been briefly described in Chapter XIII p 193 under the heading Poisonous Food

During the spawning season the quantity and the virulence of the poison of venomous fish generally increase and indeed some

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**Mollusca.**

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easy to apply proximally a tight bandage with the same precautions as mentioned under Snake Bite (see p 274)

2 *Neutralize the Poison Locally* — This should be done by opening the wound and letting it bleed while washing it with 1 per cent solution of permanganate of potash or by rubbing in crystals of the same substance and then applying fomentations and aseptic dressings

3 *General Treatment* — The pain must be relieved by hypodermics of morphia and nervous symptoms by bromides, syncopal attacks by stimulants and hypodermics of strychnine failure of respiration by artificial respiration

### Zoological Classification

The venomous fish may be classified as follows —

Subclass I Elasmobranchi

Order 3 Selachi

Suborder 3 Rau

Family Myliobatidæ

*Myliobatis aquila* Linnaeus

*Acetobatis narinari* Euphrasen

Family Trygonidæ

*Trygon pastinaca* Cuvier and other species

Subclass V Teleostei

Grade A Physostomi

Suborder 2 Cyprini siluriformes (Ostariophysi)

Family Siluridæ

*Plotosus anguillaris* Bloch 1793

*Saccobranchus fossilis* Bloch

Suborder 4 Anguilliformes (Apodes)

Family Murænidæ

*Muraena helena* Linnaeus

Grade B Physocisti

Suborder 10 Acanthoptérygii

Division 1 Perciformes

Family Acanthuridæ

*Acanthurus lurdus*

Division 7 Triglifomes (Scleroparei)

Family Triglilæ

*Trigla hirundo* Linnaeus

Family Scorpenidæ

*Synancea brachio* Cuvier and Valenciennes 1826

*retucosa* Schneider

*Scorpena grandicornis* Cuvier and Valenciennes

186

*diabolus* Cuvier and Valenciennes 1826

*porcus* Linnaeus

*Pterois antennata* Bloch

*Pelor filamentosum* Cuvier and Valenciennes 1826

Family Cottidæ

*Cottus scorpius* Linnaeus

genera—e.g. *Cottus*—are only venomous at that time. Poisonous glands being protective occur more commonly in weak and small rather than in large fish hence they are more frequent in bony than in cartilaginous fish which latter are usually of large size. Venomous fish are often conspicuous by form or colour.

**Historical.**—The history of our knowledge of venomous fish has been well written by Faust who points out that it was Aristotle the Father of Ichthyology who first established the fact that fish could produce poisoned wounds and that after his day information on this subject appears to have been very uncertain and doubts were cast upon the facts ascertained by him. Modern knowledge began in 1841 by Allman writing a paper on the stinging properties of the lesser weever (*Trachinus vipera*) which he said with all reservation was probably due to a poison gland at the base of the opercular valve. Further researches were made by Byerley in 1849 Gunther from 1864 to 1881 Corre 1865 81 Gressin 1884 and Savtschenko who produced his excellent atlas in 1886. It is to be regretted that the following names are not mentioned:—

effects still require considerable research.

**Effects of the Poison.**—As far as investigations have gone it appears as though the venoms of the different fish only varied quantitatively and not qualitatively. The effects are local and general. The local effects consist in painful sensations swelling of the part which may spread over the whole limb suppuration and even gangrene. The general symptoms appear to be due in the first instance to the action of the venom on the central nervous system which shows itself in excitation and later insensibility and paralysis. An action on cardiac muscle has also been recorded. In man death has been known to occur from wounds of *Syngnaccia brasiliensis*.

**Persons Chiefly Affected.**—The persons chiefly affected by the venom are cooks and fishermen but the latter are well aware of the danger.

**Diagnosis.**—Diagnosis is to be obtained by the history of the injury.

**Prognosis.**—This is usually good but prompt treatment is needed.

**Treatment.**—The treatment of a poisonous wound due to a fish must be based upon the following principles—

- 1 Prevent as far as possible the poison entering the general circulation
- 2 Neutralize the poison as far as possible locally
- 3 Treat the general symptoms

1 **Prevent the Poison entering the General Circulation.**—The wound will usually be on an arm or a leg and therefore it will be quite



poison and possesses an anterior convexity, but are easily moved formed by the mucosa of the palate. The poison collects between the teeth, and simply flows down them into the wound.

The venom has not been studied, except so far as to show that it has some digestive action. The effect on man is not certainly known, but is supposed to lead to syncope.

Another fish the bite of which is much dreaded, is *Tetrodon fluviatilis* which is found in the waters of Indo China, and frequently attacks the natives, especially children.

## CLASS II.

### FISH WHICH POISON BY MEANS OF STINGS ASSOCIATED WITH POISON GLANDS

Bottard classifies the poison apparatus of this class into three groups —

- 1 Apparatus entirely closed and therefore before the poison can escape a membrane must be ruptured —
  - Synanceia brachio verrucosa*
  - Plotosus anguillaris*
- 2 Apparatus partially closed —
  - Thalassophryne reticulata, maculosa*
- 3 Apparatus in more or less direct communication with the exterior —
  - Trachinus vipera draco radiatus araneus*
  - Cottus scorpius bubalis gobio*
  - Callionymus lyra*
  - Scorpena porcus scrofa*
  - Pterois antennata*
  - Pelor filamentosum*
  - Acanthurus luridus*
  - Uranoscopus scaber*
  - Trigla hirundo*

The poison glands are generally placed at the base of the dorsal or anal fins or under spines on the operculum. The gland communicates with one or more rays of the fins. The barbs may be —

1 Grooved but having the groove converted into a canal by a membrane which must be ruptured for the poison to escape

2 Canalized by channels which lead to apertures near its tip

This arrangement of the rays agrees with the types of poison apparatus mentioned above.

## Division 8 Blennioformes

## Family Trachinidae

*Trachinus draco* Linnaeus  
*viper* Cuvier and Valenciennes  
*radiatus* Cuvier and Valenciennes  
*aranensis* Risso

## Family Uranoscopidae

*Uranoscopus scaber* Linnaeus

## Family Callionymidae

*Callionymus lyra* Linnaeus

## Family Batrachidae

*Batrachus* *tau*

*grunnen* Bloch

*Thalassophryne eticulata* Günther  
*maculata* Günther

powerful spines

## Geographical Distribution

The geographical distribution of venomous fishes in temperate and tropical seas —

	<i>Teleostomi</i>
<i>Plotosus anguillaris</i>	Indian Ocean
<i>Muraena helena</i>	Mediterranean
<i>Acanthurus luridus</i>	Tropical Atlantic
<i>Trigla hirundo</i>	English Channel
<i>Syngnathus brachio</i>	Tropical Pacific
<i>verrucosa</i>	Indian Ocean
<i>Soropina grandicornis</i>	Waters of the Antilles
<i>diabolus</i>	Indian Ocean and tropical Pacific
<i>porcus</i>	Mediterranean
<i>Pterois antennata</i>	Seas of the Indian and Equatorial Oceans
<i>Pelor filamentosum</i>	Waters of Isle de France
<i>Cottus ophiops</i>	Seas of Europe, Asia and North America
Trachinidae	Waters of Europe
<i>Uranoscopus scaber</i>	Mediterranean
<i>Callionymus lyra</i>	Waters of France
Batrachidae	Waters of Tropical America and India

Leaving however the zoological classification we will consider the venomous fish according to Bottard's classification excluding Class III

## CLASS I

## FISH WHICH POISON BY THEIR BITE

The type of this class is the genus *Muraena* all of which possess powerful teeth capable of inflicting severe bites. According to Calmette there are more than one hundred species in tropical and subtropical seas. *Muraena helena* L. is found in the Mediterranean and *Muraena moringa* Cuv. in the tropical Atlantic.

The poison apparatus consists of a pouch lying above the covering of the palate lined with epithelial cells secreting the venom. This pouch is capable in larger species of holding about 4 c.c. of

poison and possesses three or four strong conical teeth curved with an anterior convexity. These teeth are not grooved or channelled but are easily moved and erected and are enclosed in a sheath formed by the mucosa of the palate. The poison collects between the teeth and simply flows down them into the wound.

The venom has not been studied except so far as to show that it has some digestive action. The effect on man is not certainly known but is supposed to lead to syncope.

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*Trachinus vipera  
liraco  
radiatus  
araneus*

*Cottus scorpius  
bubalis  
gobio*

*Calonymus lyra  
Scorpaena porcus  
scrofa*

*Pterois antennata  
Pleur filamentosum  
Acanthurus luridus  
Uranscoelus scaber  
Trigla hirundo*

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1 Groove  
membrane

2 Canalized

This arrangement of the rays agrees with the types of poison apparatus mentioned above.

## Section 1—Poison Apparatus Entirely Closed

The apparatus is connected with the dorsal fin and is passively defensive—that is to say the fish cannot eject the poison unless the barb is broken.

EXAMPLES—*Synanceia* (*brachio* and many other species) *Plotosus arab*

*Synanceia* (*brachio* and many other species)—This fish called rapau de mer in Reunion laffe in Mauritius ikan Sutan in Java and nohu in Tahiti is distributed through the waters of

enter the skin and the poison is pressed mechanically into the wound. The venom is limpid bluish and slightly acid. The



FIG. 27.—*Synneca verrucosa* SCHNEIDER  
(From Savtchenko's Atlas of Poisonous Fish.)

symptoms are severe pain spreading up the limb. The sufferer becomes violent, throwing himself from side to side and even asking to have his foot cut off. Sometimes syncope and death may take place. In other cases abscesses and symptoms of blood poisoning may be noted. The skin surrounding the wound becomes bluish and may slough in which case recovery takes a very long time to be completed.

*Plotosus anguillaris*—This fish called machira in Reunion and Mauritius sanbilang in Malay koormat in Abyssinia is found in the waters of India of the Seychelles and other places mentioned above. The poison apparatus is connected with the dorsal fin and the conditions which bring about the wound together with the symptoms resemble those of *Synanceia*.

*Saccobranthus fossilis*—This fish is found in the waters of India and Ceylon and wounds caused by its pectoral fins are much dreaded by the natives as they produce severe inflammation and even tetanic symptoms.

*Scorpaena scorpa*—In this fish the poison apparatus is said to exist not merely in the dorsal fin but also in the operculum

In the dorsal fin the first three rays which project about one third of their length beyond the membrane of the fin are grooved posteriorly by three channels which are converted into canals by a fine membrane and communicate with the poison gland

On the operculum there are three small spines of which the

und may result from

The action of the poison has been studied by Pohl Brunton and Briot Brunton describes the effects of the poison as being exactly the same as that of *Trachinus draco* and Pohl thinks that the poison acts on the heart in the same manner as he describes for *Trachinus*



FIG. 29.—*Co tus scorpa* LINNÆUS  
(From Savitschenko's Atlas of Poisonous Fish)

On the other hand Briot thinks that it is quite different from

or *Scorpaena* which affected the person His experiments on animals with both fish appear however to have caused the same symptoms He pressed the spines of the poison organs against the hind limb of a guinea pig or a rat In a few minutes the animal began to suffer pain and twitching in the injured limb followed by tremors and convulsions (if disturbed) and later by death from collapse

The nature of the system in human beings but reference to reader that he is

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*Synaesa* (*Trachis* and many other species)—This fish called rapau de mer in Réunion laffé in Mauritius ikan Satan in Java and nohu in Tahiti is distributed through the waters of the Indies Cochinchina New Caledonia and the Pacific Ocean.

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FIG. 7.—*Synaesa veryi* O. SCHNEIDER  
(From Savitschenko's Atlas of Poisonous Fish.)

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phlegmonous and gangrenous symptoms so severe that the arm had to be amputated

*Acanthurus luridus*—*Acanthurus luridus* possesses a poison apparatus connected with the dorsal and anal fins like that of *Scorpena*

*Elasmobranchii*—Associated with the poisonous fish of the *Teleostei* must be placed those of the *Elasmobranchii* of which the sting rays (*Trygonidæ*) and eagle rays (*Myliobatidæ*) alone produce toxic symptoms by blows with the tail which carries a spine. These Rays are found all over the world and we have received information as to their effects from persons who have been in British Guiana in Australia and in Ceylon in which island stings are well known on the west coast particularly about Dutch Bay

Dr Crevaux has studied Rays from the Orinoco and has shown that their barbs are canalized and the canals connected with poison reservoirs. This poison is said to be so severe as to be able to kill a man in forty eight hours. The symptoms of *Aetobatis narinari* called the Bishop ray and of *Trygon pastinaca* (from Japan) are violent pain a tendency to syncope, with locally a rapidly forming swelling which soon becomes the seat of a violent inflammation and even at times gangrene. The symptoms of the sting as observed by us in Ceylon are local pain and swelling. The general symptoms are not severe. *Trygon sephen* and *T walga* Mull and Hen are known in Indian waters.

A large number of these *Elasmobranchii* have not got special poison glands and the venom must come from the ordinary skin glands.

### Amphibia

Toads and salamanders have been celebrated for ages as venomous animals the poison being found in their parotid glands and skin. In toads Faust has shown that there are two poisons—(1) an acid bufotain and (2) a neutral body bufonin the former being the more active. It is as a rule scarcely toxic to man only irritating the mucous membranes especially the conjunctiva but

is here

According to Vulpian and Capurelli *Triton cristatus* (Laur) gives a creamy secretion from the glands of the skin at times which is poisonous to many animals but the chemical nature of which is not known

The symptoms of an attack may be described thus —

A sharp prick is felt as the spine enters the skin and this is followed in a few minutes by burning and itching which shortly become stabbing pains increasing in violence and passing up the limb. The sufferer now lies down and writhes and cries in agony while sweat breaks out on his brow and flashes of light pass in front of his eyes. He begins to feel a sensation of suffocation and puts his hand to his throat and heart while the pulse is felt to be intermittent. Presently he loses sight of the bystanders and in a little becomes delirious crying out and suffering from convulsions. This condition may lead to collapse and death or after lasting several hours may gradually subside but the convalescence is slow and the patient may take several months to recover.



FIG 30 — *Scorpaena diabolus* CUVIER AND VALENCIENN  
(From SAVITSCHENKO'S "Atlas of Poisonous Fish")

The local conditions show at first merely the prick of the spine but the aperture may be pigmented by the covering membrane already alluded to. The puncture does not bleed but the skin around for about  $\frac{1}{2}$  inch is whiter than usual and outside this red

ing gangrene ensues which necessitates amputation. In milder cases the part remains painful and swollen for about three days before recovery.

*Pterois antennata* — The poison apparatus is connected with the dorsal fin and is said to resemble that of the Scorpænidæ.

*Pelor filamentosum* — In this fish the poison apparatus is connected with the dorsal fin and resembles that of *Pterois* and *Scorpaena*. With regard to *Pelor japonicum* Cu and Val S. Heube says that he knew of a case in which a sting on the thumb resulted in



The results were as follows —

	minutes
	35 minutes
	minutes
	minutes
	minutes
	minutes

Next they tried two MLD with the following results —

	hours
	hours

From these experiments they conclude that snake venom contains a neurotoxic principle which is the chief poison and which

This neurotoxic substance has been shown by Rogers in the cobra and the *Hydrophulæ* to attack the respiratory centre in the medulla the respirations becoming both fewer in number and less in amplitude minute by minute until they cease if sufficiently large doses are administered but when smaller doses are given there is at first a temporary stimulation

Further he shows that the neurotoxic substance can paralyze the end plates of the phrenic nerves in the diaphragm shortly after the failure of the respiratory centre in the medulla

The blood pressure does not appear to be affected by the poison and in fact the circulation can be kept going for a long time after cessation of breathing if artificial respiration is resorted to—a fact first shown by Brunton and Fayrer

On the other hand with regard to viperine snakes—e.g. *Vipera*

### Crotalus poisoning

There are thus several neurotoxic elements in snake venom of which two great groups can at present be provisionally described

(1) Colubrine neurotoxic element acting upon the—(1) Respiratory centre in the medulla (2) the end plates of the phrenic nerve

(2) Viperine neurotoxic element acting upon the vasomotor centre in the medulla

It has been stated that the effects of the venom of *Hoblochela* is

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## Amphibia

- CALMETTE (1907) *Les Venins* pp 328 332  
 FAUST (1900) *Die Tienschen Gifte* p 210  
 WELLS (1906) *Chemical Pathology* pp 182 183

the agglutinated red corpuscles which finally became invisible when treated with *Crotalus* venom. Feoktistow showed that a 2 per cent solution of the venom of *Vipera berus* destroyed red corpuscles in eighteen to twenty four hours. Martin has demonstrated also that the venom of *Pseudechis* in 0.1 per cent solution destroyed the red corpuscle.

Flexner and Noguchi showed that these hæmolysins must be looked upon as amboceptors which require a complement and that they obtain in the bactericidal principles found in the serum of the victim. Hence when they join with the erythrocytes and the complement they not merely produce hæmolysis but they take away the bactericidal powers from the blood. If as was found to be the case in *Necturus* they are incapable of uniting with the complements they are incapable of damaging the bactericidal properties of the serum for it was found that hæmolysis was but slightly produced in the blood of *Necturus* and then only after long periods and at the same time the serum of *Necturus* did not lose its bactericidal effects to *Bacillus coli communis* and *B. typhosus*.

The hæmolysins have been further studied by Kyes who showed that in some animals the venom alone could hæmolyze the washed red cells whereas in others it could not do so until some fresh serum was added. In the first class came man, dog, rabbit, guinea pig and horse and with reference to these he came to the conclusion that the complement was contained in the red blood cell itself—i.e. was an endocomplement. In a further research with Sachs he shows that this endocomplement is attached to the stroma of the red cells. Further they conclude that it is the lecithin of the stroma which acts as the complement and support this by experiments showing that lecithin prepared from the yolk of an egg can act as a complement for the venom and dissolve cells which are not affected by the venom alone. They look upon the fatty acid radical of the lecithin as being probably the active agent.

In the second class—viz. those animals whose erythrocytes are not affected by venom alone without the presence of serum—come

to produce  
l by venom  
guinea pig  
amboceptor

in the centre of the cell. The cells are unequally attacked. Those around the central canal are most severely injured while the motor cell

He says †

as those

very like

and vascular changes are absent

As to the cause of this cytotoxicity it would appear from the researches of Flexner and Noguchi that it is probably due to the union of an endocomplement (capable of being neutralized by calcium chloride and therefore not lecithin) in the nerve cell with an amboceptor in the venom. This neurotoxin according to Ehrlich's denomination is neurotropic—†e unites only with nerve cells—and monotropic—†e has affinities for one tissue only.

These facts have been demonstrated by Flexner and Noguchi in cobra venom by first treating it with erythrocytes to remove the hæmolysin and then heating it to destroy the hæmorrhagin thus leaving only the neurotoxic principle to act on the animal.

They found that after cerebral injection the appearance of the nervous symptoms was almost immediate while with other methods of injection the development was more gradual.

The first effect of the neurotoxin was irritation (convulsions) and the final paralysis. By cobra venom death was caused by respiratory paralysis. Intracerebral injection of a viperine venom like that of *Crotalus* however caused but slight symptoms.

They further found that the venoms of *Ancistrodon piscivorus* Lacep and *A. contortrix* L occupied an intermediate position between the colubrine and viperine venom containing both neurotoxins and hæmolysins in considerable quantities. From these experiments it was possible to classify venoms into the three classes given above.

2. AGGLUTININS ← Agglutination of the red cells was first described by Weir Mitchell and Reichert and that of the leucocytes by Halford and Ralf.

The reaction can be studied by adding a 0.01 per cent to 10 per cent solution of dried venom in normal saline to washed corpuscles suspended in normal saline.

The time taken for the reaction to occur is as follows:

considerably altered. Agglutinins are destroyed by heating the venom to 70°-80° C.

The agglutinins for the red cells appear to be the same substances as those for the white and in both cases they appear to

Hence Kyes concludes that the hæmolytic element of

while cobra venom will unite with Calmette's antitoxin *Vipera russelli* will not. *Bungarus* and *Naja bungarus* the cobra and *Lachesis* and *Crotalus* do not.

Noguchi investigated lecithin and found it to be by an inert substance and further that certain oleic compounds oleic acid itself would act as venom activators.

He found that the addition of oleic acid or its soluble a non activating serum in the ratio which corresponds to percentages of fatty acids and soaps contained in some of activating sera makes it highly active to venom. In the serum of a dog however he found that there was a lecithin compound acting like free lecithin. He further found that classes of activators could be differentiated from one another a calcium chloride which annulled the first group but was against the lecithin. Non activating sera do not contain lecithin compound and other lecithides—*e.g.* lecithin albumin powerless.

When serum is heated the non coagulated portions lecithin activator as described by Kyes which is identical with Chabrié's albumon but this does not exist preformed in sera and is due to the high temperature altering other lecithides into albumon.

He further found ovovitellin to be one of the best lecithin activators. He finally came to the conclusion that the red corpuscles (man etc.) are acted upon by venom others (ox etc.) are not depends solely upon the amount of fatty acids and perhaps also of soaps and fats contained in the corpuscles to the stroma of which they are attached. The larger corpuscles are plentifully in corpuscles easily affected and in small corpuscles those not easily affected.

The position at the present moment is therefore that venom produces hæmolysis by its amboceptors uniting with the lecithin elements contained in the sera of the majority of man

species and these are of the same nature as those in non hæmolytic sera and are attached to the stroma of the corpuscle.

Finally the reason why heated sera become active at higher temperatures after losing their activity at lower temperatures is due to the conversion of the proteid lecithides into another form of lecithide called albumon which is an activator.

For the controversy between von Dungern Coca supports the former view and Kyes on the other refers to the

believe that the serum merely frees the lecithin which acts as the true complement

In (2) the lecithin being free is able to combine with the antibody and cause the hæmolysis. They further show that if a blood cell is treated with cobra venom, the lecithin is destroyed and the cell is not available.

lecithin is not available

Kyes then went a step further and mixed 40 c.c. of a 1 per cent solution of cobra venom in a 0.75 per cent salt solution with 20 c.c. of 20 per cent solution of lecithin in chloroform and then centrifuged the mixture. The result was the separation of chloroform and water into two layers from the former the cobra venom lecithin could be precipitated by the addition of pure ether. This body possessed hæmolytic but no neurotoxic properties which entirely remained in the water. Thus Kyes clearly supports the fact first pointed out by the late Dr Myers that the neurotoxic and hæmolytic

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achesis

1	<i>L. mullensis</i> Gunther	(2) <i>L. lanceolatus</i> Lacep	(3) <i>L. flavoviridis</i> Shaw	(6) <i>Naja fasciatus</i> Schm
2				lecithin destroy

and cells and except in the first and the third the absolute quantity of the poison is the same as that mentioned just above.

That of *Lachesis anamallensis* Gunther is however twenty five times weaker and *L. lanceolatus* Lacep ten times weaker this being due to the fact that the former forms only one twenty fifth and the latter one-tenth the usual quantity of lecithin.

(d) *Other Cytolysins*—Flexner and Noguchi have found that the amoebocytes in venom can act upon a number of the cells of the body—e.g. liver kidney, testicle and ovary—causing lysis the complement being probably either endocellular or in the lymph stream. The nature of the complement is however quite unknown at present but is probably different on the red cells as its activity some time. The histological changes particularly in the microscopical appearances in the liver and kidney. The liver shows necrosis and fatty degeneration of its cells and as a reactionary process leucocytic infiltration around the bile passages. In the kidney the glomeruli show intense congestion of their capillaries often associated with ruptures of their walls and hæmorrhage into Bowman's capsule. The cells of the tubules are necrotic and detached filling the lumen. The whole organ is congested and there may be interstitial hæmorrhages. The spleen is but slightly affected.

4 ANTIHÆMOLYSINS—Weir Mitchell and Stewart have shown that if crotalus venom is added to red corpuscles in a certain degree of concentration no hæmolysis takes place. This fact has been confirmed by Myers and Stephens for the cobra and Lamb for *Lipera russellii*.

Noguchi believes that the action is due to venom having the power in certain cases of precipitating the outer layer of the hæmoglobin while in other cases this does not take place.

5 ANTIBACTERICIDAL SUBSTANCES—Weir Mitchell drew attention to the fact that venoms rapidly decomposed the bactericidal power of the serum.

Flexner and Noguchi also investigated this action and came to the conclusion that—

(1) All venoms when used in suitable quantities destroy the bactericidal properties of many normal sera.

(2) The manner of this destruction consists in the fixation of the serum complements by the venoms.

(3) Venoms have no action upon the intermediary bodies of the serum.

6 FIBRIN FERMENT—A fibrin ferment has been shown by Martin to be present in the venoms of the viperidæ and also of some of the colubridæ. In the former it is the active agent which causes the vascular clotting in small animals with convulsions and sudden death. This coagulative substance has been shown by Barratt to be a thrombin.

7 ANTIFIBRIN FERMENT—An antifibrin ferment—that is to say the

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be made to the original papers given in our list at the end of this chapter

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dehis venom and says that for the first fifteen minutes he could see no change in the white cells but they exhibited no amoeboid movements. At the end of this time the nuclei in some of them were very distinct as if fixed by acetic acid. They then became intensely granular and soon began to swell and their outlines to grow less distinct until they disappeared leaving only a small

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cytes. They found cobra poison much more vigorous than that of crotalus. As to the leucolysins they proved that they were separate from the hæmolysins by treating washed red corpuscles with copperhead venom until the supernatant fluid after centrifugalization ceased to have any effect on red cells. This fluid was then brought into contact with leucocytes when lysis without agglutination took place. If however washed leucocytes were treated first the supernatant fluid was found to be actively hæmolytic. They therefore concluded that the hæmolysins were distinct from the leucolysins but that the agglutinins were probably the same.

We are not aware of further researches as to the nature of the leucolysins though obviously such researches are required.

(c) *Hæmorrhagin*—Weir Mitchell and Reichert by observing a mesentery moistened with crotalus venom came to the conclusion

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hæmatotoxic properties (hæmolysins and agglutinins) in the venom and that as the latter can be eliminated without any apparent loss of toxicity it must be due to the neurotoxins.

The toxic principle lost by heating to 75° C. Flexner and Noguchi called hæmorrhagin. They studied its action in the mesentery by injecting the venom into the peritoneal cavity or placing a minute particle of the dried poison on the exposed mesentery and then removing specimens which they fixed in Zenker's fluid cut into sections and stained with hæmatoxylin and eosin. They found that the extravasation of blood took place not by diapedesis but through actual rents in the walls. These rents are not simple ruptures but are apparently due to a cytolytic action upon the endothelial cells of the capillaries and the walls of the small veins.



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Microscopically fatty degeneration of the liver and kidney and necrosis of hepatic and renal epithelium are to be seen as well as round celled infiltration along bile ducts which is probably due to excretion of poison

Turning now to the colubrine snakes which resemble the cobra there are observations upon *Naja haje* *N bungarus* *Bungarus candidus* *B fasciatus* and the *Hydrophidæ*

*Naja haje* Linnæus—Calmette records that Dr Deschamps observed a case of this bite in Senegal in which the snake bit the patient in the forehead. Almost immediately great weakness accompanied by nausea and pains in the head and neck set in. Locally two raised areas were seen around which the tissue was œdematous. Cold sweats occurred. The œdema spread to the face dyspnœa appeared and the pulse became small and intermittent paralysis set in and the patient became comatose but recovered on treatment with antivenene.

*Naja bungarus* Schlegel—*N bungarus* the hamadryad is by far the biggest of the Indian poisonous snakes. Rogers finds its venom very like that of the cobra producing paralysis and death from failure of respiration while the heart continues to beat for a time.

There is no intravascular clotting and the hæmolytic action is very slight but the phrenic nerve plates are paralyzed. If small doses are given the respirations are increased.

We are not acquainted with the symptoms exhibited by an undoubted case of this bite in a human being.

*Bungarus candidus* Linnæus—As to *B candidus* L. the commonest of all Indian snakes there is not much to say except that all researches (Fayrer Wall and Rogers) indicate that its venom is almost exactly the same as that of the cobra but is slightly more virulent.

Cases of this bite are common. In brief the symptoms are—a sense of tightness across the chest with paralysis particularly of the muscles of the face deglutition and phonation. The conjunctivæ are suffused the pupils dilated the pulse and respirations quickened the temperature is normal and the local signs are not marked.

Coma and convulsions precede death which is due to failure of respiration. Congestion of the meninges and brain and liquid blood are the principal signs found post mortem.

*Bungarus ceylonicus* (Gunther)—*B ceylonicus* the Ceylon krat or carawalla has been reported by Green to have killed a man in twelve hours. The man was bitten on the left foot at 4 a m and felt quite well till 5 30 a m when he felt drowsiness which increased till 10 a m when he could hardly swallow and was very

Thus Mitchell and Reichert long ago showed that muscle fibres at the site of the bite were quickly softened by crotalus venom and Flexner and Noguchi have shown that gelatin is liquefied by both crotalus and cobra venoms but that coagulated proteids are not acted upon. Microscopically the muscular fibres at the site of the bite are seen to have undergone necrosis and degeneration and later a polymorphonuclear leucocytic infiltration may be noted.

9. **CARDIAC AND VASCULAR TONIC**—Sir Lauder Brunton and Sir Joseph Fayrer showed that the poisons of the cobra had a stimulant effect on the heart and that the circulation could be kept going for a long time after complete failure of breathing if artificial respiration is kept up—a fact of considerable importance if any antidotal treatment is available.

It is not clear whether this is due to direct action on the muscles of the heart but it appears from Rogers' experiments that the effect on the bloodvessels is due to local action on the arterioles. This effect is produced by the cobra and *L. spera russellii* venom.

### Entry of the Venom into the Body

The poison the characters of which have been described is introduced into the body of man or that of an animal usually by the snake biting the skin and injecting the poison either subcutaneously or into a vessel. In the former case it will soon reach the blood stream and be distributed to the different parts of the body.

The effect of entry into a bloodvessel is to produce immediately the signs of the poisoning whereas in the subcutaneous tissue it may take some time and even be modified especially in the viperine type.

The quantity injected by a cobra according to Acton and

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If the venom falls on the conjunctiva it is readily absorbed and symptoms of poisoning will ensue or an acute inflammation be set up.

Taken by the mouth the venom of the colubridæ is harmless provided there are no cracks or abrasions and is destroyed by the saliva and pancreatic juice. Viperine venom on the other hand causes gastritis gastro-intestinal hæmorrhage and even death without the appearance of the usual symptoms, a fact known to the natives of Ceylon with regard to the venom of *L. spera russellii*.

### Minimum Lethal Dose (M.L.D.)

The minimum lethal dose varies with the species of snake, the condition of the snake and with the species of the victim.

*Naja flava* and *N. nigricollis* & *tripudians* *Echis carinata* with perhaps the addition of *Naja melanoleuca* and *Vipera russelli*, and it is possible that further observations will show that all the Co's

that of the cobra in the following points —

- 1 They were more toxic
- 2 They were much less hemolytic and hence caused no blood-stained effusion at the site of the injection
- 3 They did not affect the coagulability of the blood therefore the poison is almost purely neurotoxic

There is at first an excitation of the nervous system leading to a feeling of activity and vivacity, which however soon passes off. The earliest signs of distress begin with difficulty of articulation and feeling of stiffness in the body and of suffocation. The stiffness in the muscles increases and occasional spasms occur while signs of gastric irritation with vomiting appear. Convulsions and death may ensue after a day or so. The local signs may be slight.

**Viperine Venom** — *Vipera russelli* Shaw the *n. palonga* of Ceylon is a good example of this type of venom. Experiments show that a dog bitten by one of these was convulsed and die within five minutes. The cause has been shown by Rogers to be intravascular and is best marked in the portal vein and then only

sick Paralysis affected his legs and he became cold. Artificial respiration was now resorted to but at 2 p.m. he became very

tion the blood is coagulated and there is paralysis and sometimes convulsions

Chronic cases on the other hand last for days during which

rather deeply stained plasma in which are scattered dust like granules the remnants of the Nissl bodies many cells show vacuolation of the plasma and some are reduced to mere outlines (ghost cells)

Rogers experiments tend to show that even in injecting large doses the blood may not clot and that the animal may be killed as in cobra venom by direct action of the poison on the respiratory centre and also on the nerve endings of the phrenics so that the venom appears to be a mixture of a viperine with a colubrine poison

The only authentic case of human poisoning by *B. fasciatus* is mentioned by Gayler in which there was tingling sensation and later pain at the seat of the punctures with some swelling all of

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patient becomes unable to stand signs of prostration accompanied by vomiting appear The circulation begins to fail the heart's action becomes weak the extremities cold and the skin blanched while the respiration after a preliminary excitation becomes slowed Coma now intervenes the sensations being diminished and the pupils dilated and death results from failure of the respiration preceded sometimes by convulsions The heart continues to beat after the circulation has ceased

*Elaps fulvus* the harlequin snake which is found in the eastern parts of the Southern United States causes great local pain followed in one hour by drowsiness unconsciousness and collapse lasting until death or for a day or so if the patient recovers Death usually ensues in about twenty four hours after a bite and in persons tending to recovery the danger of death is not escaped until three to four days have passed away as the symptoms tend to recur periodically

*Spitting Snakes*—The known spitting snakes—the snakes which can project their venom to a distance—are *Sepedon haemochates*

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In addition to these acute cases there are also chronic cases in which the local symptoms are more or less extensive subcutaneous hæmorrhages around which there is much œdema. This hæmorrhage may be absorbed or the area may slough or an abscess or even a spreading gangrene may ensue.

The general symptoms are rapid emaciation profound anemia and lethargy and in some cases hæmaturia and a discharge of blood from the bowel.

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hurried respirations and later stuporous. The skin is clammy and covered with a cold sweat while the swelling and discoloration spreads considerably reaching the trunk. In due course convulsions set in and death ensues from failure of the circulation. The post mortem reveals congestion of the meninges and lungs with fluid blood and nothing else of importance.

*Bitis arietans*—The puff adder poison was found by Rogers to work in much the same manner as that of *Vasera russellii* but not

nous snake and the effects

was much local swelling passage of blood in the urine, feces and vomit, elevation of temperature and death from exhaustion due to loss of blood on the ninth day. Martin and Lamb record another case in which there was much swelling of the bitten part due to exudation of liquid blood. Pain and tenderness were felt along the nerves together with anæsthesia, extreme restlessness with cold and clammy ex-

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Reichert and by *Lachesis* which latter has been studied by Rogers in India.

effect on the respiratory centre and the phrenics are not paralyzed. The heart goes on beating after respiration ceases but is slightly weakened. Post mortem examination shows hæmorrhages into the peri- and endocardium and into the peritoneum and pleura but not in the brain or the medulla while the whole portal system is much congested.

Rogers placed a loop of small intestine in an oncometer and found that fall of blood pressure was associated with a vaso-dilatation of the portal system in which the blood was not clotted. This vaso-dilatation he considers may possibly be due to the action of the venom on the vasomotor-centre in the medulla and thinks that he is supported in his theory by the appearance of Traube-Hering curves in his blood pressure tracings. Pearce notes acute glomerular lesions due to the endothelialytic body.

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*The Hydrophiidæ*—Rogers experimented with *Enhydrya talakadien* Boie *Distrya cyanocincta* Daud and *Hydrophis cantoris* Gthr and came to the conclusion that their venom only differed from that of the cobra in the following points—

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In addition to these acute cases there are also chronic cases in which the local symptoms are more or less extensive subcutaneous hæmorrhages around which there is much œdema. This hæmorrhage may be absorbed or the area may slough or an abscess or even a spreading gangrene may ensue.

The general symptoms are rapid emaciation profound anæmia and lethargy and in some cases hæmaturia and a discharge of blood from the bowel.

Rogers has carefully investigated these symptoms and has pointed out that there is a remarkable fall of blood pressure due to vaso dilatation of the portal system caused by action of the poison on the central and not the peripheral vasomotor apparatus the heart muscle being unaffected. Lamb and Haun showed that in chronic cases there is deficiency in the blood coagulability

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Weir Mitchell and others have carefully collected the symptoms following this bite. In most instances the bite is painful and the part becomes swollen and discoloured while both the pain and the swelling increase steadily. The swelling is due to the effusion of blood. Very seldom is there any lymphangitis or enlargement of the lymphatic glands. Vesication sloughing and

animal. Under exceptional circumstances it is recorded that the symptoms did not begin till about thirty minutes after the bite but usually the length of time is only a few minutes. The person feels extremely faint or complains that his lower limbs are not able to support him. There is no primary stimulating effect like that mentioned in other venoms. The patient staggers or falls, cold sweats bathe the surface of the skin and nausea and vomiting occur. The pulse is rapid and feeble, the expression anxious and according to Mitchell in a few cases the mind may be slightly disturbed but this may be largely due to fear. If the patient does not die at this stage the local symptoms mentioned above become very pronounced and signs of general blood poisoning show themselves and often lead to death.

The post mortem reveals that the brain is normal but congested and somewhat œdematous, the lungs are somewhat congested and full of red frothy mucus. The peritoneum and the mucosa of the stomach and small intestines may be intensely congested and infiltrated with serum. In some cases the blood is coagulated in some it is fluid. The local swelling is due to serous exudation. The chief features of the post mortem are vaso dilatation of the portal system and fluidity of the blood.

*Lachesis*—This snake shows the same symptoms as *Crotalus* but it has some effect upon the respiratory centre causing quickening of respiration before the slowing begins. The phrenics are not paralyzed. Moreover it is possible that the first quick fall of blood pressure observed is due to action of the poison on the heart but the subsequent steady fall is due to the action on the vasomotor

cold sweats<sup>1</sup> come on just before death. Blood has been observed in the urine and fæces.

**Excretion of the Poison.**

It is believed that the poison leaves the body principally by the kidney and to a less extent by the mucosa of the stomach the

is some direct proof of the  
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when injected hypodermically into a pigeon caused death  
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**Immunity**

Having considered the chemical composition and physiological action of the venom the next point to be discussed is immunity to its action

Ancient and modern peoples have equally held the belief that a person who

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Acquired immunity is said to occur among natives especially snake

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In 1897 Calmette showed that by repeated inoculation of venom heated to 80° C a certain amount of resistance was produced in animals. In 1894 he made researches on the venom of the cobra and about the same time Phisalix and Bertrand investigated that of the viper and showed that animals vaccinated with venom developed a true immunity and those inoculated against the cobra venom were able to resist mortal doses of *Vipera Bungarus*, *Cerastes*, *Naja haje* and *Pseudechis* venoms. Later they showed

Weir Mitchell and others have carefully collected the symptoms following this bite. In most instances the bite is painful and the part becomes swollen and the swelling increase effusion of blood. Very enlargement of the lymph gangrene may result if life. If on the other hand the amount of poison injected is inconsiderable the swelling declines and the pain disappears very quickly. Constitutional symptoms are said after the bite but this is hardly animal. Under exceptional circumstances symptoms did not begin till about thirty minutes after the bite but usually the length of time is only a few minutes. The person feels extremely faint or complains that his lower limbs are not able to support him. There is no primary stimulating effect like that mentioned in other venoms. The patient staggers or falls cold sweats bathe the surface of the skin and nausea and vomiting occur. The pulse is rapid and feeble the expression anxious and according to Mitchell in a few cases the mind may be slightly disturbed but this may be largely due to fear. If the patient does not die at this stage the local symptoms mentioned above become very pronounced and signs of general blood poisoning show themselves and often lead to death.

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poisonous snake

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Acquired immunity is said to occur among natives especially snake charmers and Europeans who have been bitten several times by snakes

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The first scientific attempt to produce an artificial immunity was made by Sewall in 1887 when by repeated small injections he raised the resistance of persons so high that they were able to resist ten times the minimum lethal dose of the venom of a *Crotalus*. Kanthack also produced a partial immunity to cobra venom in 1891 Kaufmann a little later obtained a similar result with the French viper

In 1892 Calmette showed that by repeated inoculation of venom heated to 80° C a certain amount of resistance was produced in animals. In 1894 he made researches on the venom of the cobra and about the same time Phisalix and Bertrand investigated that of the viper and showed that animals vaccinated with venom developed a true immunity and those inoculated against the cobra venom were able to resist mortal doses of *Vipera Bungarus*, *Cerastes*, *Naja haje* and *Pseudechis* venoms. Later they showed

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the pulse feeble and rapid respiration is normal a slight fever is not infrequent. Coldness of the extremities with dyspnoea and cold sweats come on just before death. Blood has been observed in the urine and fæces.

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it to see whether it is poisonous or not especially to see whether there are fangs and if non poisonous the patient's mind can be relieved at once

### Prognosis

It appears that only a relatively small percentage of persons bitten by snakes (supposed to be poisonous) die C J Martin and Lamb place it about 30 per cent but much depends upon the quantity of venom injected and the rapidity with which symptoms develop The recoveries from full doses of echus venom in untreated cases is about 40 per cent and of cobra venom only 3 to 4 per cent Personally we believe that in most cases if treatment has been prompt and the symptoms appear slowly, the outlook is by no means hopeless

### Treatment.

Snake poisoning consists in the hypodermic or intravascular injection of a series of poisonous principles which act chiefly upon the nervous system and the blood

The virulence of the poisoning depends upon the ratio of the quantity of the poison injected to the size of the animal The same quantity of poison will therefore have a more serious effect upon a child than upon an adult The less the quantity of poison which gets into the general circulation the less the symptoms, hence the first indication for treatment is to prevent the passage of the poison as far as possible into the circulation

The second indication for treatment is to neutralize the poison  
 The third indication is to treat

the General Circulation—In  
 into the general circulation

- (1) Stoppage of the flow of blood and lymph from the affected area
- (2) Free opening of the poisoned area
- (3) The neutralization of the poison locally

(1) *Stoppage of the Flow of Blood and Lymph from the Affected Area*—A person is usually bitten in the arm or leg, and in such a

arm or leg

Such a

gangrene

the poison

seconds and reapplied, and this must be repeated It must be kept on for at least twenty or thirty minutes

The advantages of the ligature or tourniquet have been known since ancient times but C. J. Martin has shown that its principal

locally retains the poison which therefore but slowly passes into the general circulation and may as Martin and Lamb suggest

local & proximal tourniquet

notes

Therefore the next step is to cut not merely round the apertures of the fangs but also to extend the incision along the course of the veins and lymphatics and in some cases to remove the piece of skin marked out. In this way the area of inoculation is freely opened.

(3) *Neutralization of the Poison Locally*—The next indication is to diminish the toxicity of the poison as much as possible and to do this the best remedy we at present possess is *permanganate of potash* though recent laboratory experiments have thrown some doubt upon its efficacy.

This should be used in strong solution and not as a solid as the liquid penetrates better into the interstices of the wound which should be well washed with it. For carrying out this treatment a most useful little case has been devised containing a little lancet for making the wound and permanganate crystals for making a solution. This little case only costs a few pence and can be carried without any inconvenience in the waistcoat pocket and therefore should be carried by everyone in countries where poisonous snakes abound especially when on business or pleasure in jungles or grassy places. Should there be no water available to make a solution then the crystals can be rubbed into the wound.

If the bite is on the head or trunk the incision should be made as above and the resulting wound thoroughly soaked with permanganate of potash solution (3 per cent). The subcutaneous tissue must be freely opened otherwise the permanganate may be prevented from doing its work properly.

The after treatment should be hot fomentations frequently repeated at first but if there is no marked swelling of the part these may be gradually not quickly diminished in number and finally a mild antiseptic dressing should be applied until the wound heals.

2 *Neutralization of the Poison in the System*.—We have already endeavoured to impress on the reader the following facts—



**Prophylaxis**

In the tropics Europeans and better-class natives seldom suffer from snake-bite because they do not sleep on the ground in the open or in small huts, like the lower class native. Among the latter however any prophylaxis is difficult in regions such as India because they reverence the cobra and encourage it to take up its abode in their huts.

Gardens should be kept free from jungle or long grass and gratings should be placed on the drains from bath rooms as snakes have often been found in these rooms.

Good strong boots and the puttee pattern of legging should be worn when going shooting or into the jungle. A lantern should be used when walking after dark and the small permanganate case already mentioned should be carried in the waistcoat pocket in case of accidents.

**LACERTILIA**

**FAMILY HELODERMIDÆ** Gray 1838

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Sumichrast in  
it is probable

**GENUS HELODERMA** Wiegmann 1834

*Heloderma horridum* Wiegmann 1834

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**Heloderma suspectum** Cope 1869

Cope gave the lizard the name *suspectum* because he suspected that its bite would be found to be poisonous. This lizard is found in the United States—in Arizona, Texas, Utah, New Mexico, and Southern California—where it is called the gila monster. The poison apparatus consists of glands under the lower jaw and teeth in that jaw.

symptoms—Very severe pains radiating from the part, rapid swelling, faintness, and Tr. man. ficial.

**FAMILY LANTHANOTIDÆ** Steindachner 1877

This family was formed for *Lanthanotus* Steindachner 1877 which is the type genus. It is closely related to Helodermidæ.

**Lanthanotus borneensis** Steindachner 1877

This lizard which is closely allied to *Heloderma* is suspected of being poisonous but there are as yet no proofs of this especially as the teeth are not grooved and there is doubt as to the presence of poison glands. Its habitat is Borneo but it requires reinvestigation as but few specimens are known.

**MAMMALIA** Linnæus

The mammal suspected of causing poisoning is *Ornithorhynchus paradoxus* belonging to the Monotremata.

**MONOTREMATA** Bonaparte 1837**Ornithorhynchus paradoxus** Blumenbach 1800

As is well known, this animal is found in Australia. These pursuits are the venom is an



**Heloderma suspectum** Cope, 1869

Cope gave the lizard the name *suspectum*, because he suspected that its bite would be found to be poisonous. This lizard is found in the United States—in Arizona, Texas, Utah, New Mexico, and Southern California—where it is called the 'gila monster'. The poison apparatus consists of glands under the lower jaw and teeth in that jaw.

The poison gland in this species lies on either side of the lower jaw and

**FAMILY LANTHANOTIDÆ** Steindachner 1877

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**Lanthanotus borneensis** Steindachner 1877

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**MONOTREMATA** Bonaparte 1837**Ornithorhynchus paradoxus** Blumenbach 1800

As  
have  
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albus

lives in the red cells of the blood. These latter may be subdivided into periodical parasites which only spend a portion of their life-history within a host and permanent which are parasitic throughout the whole life cycle. As an example of the former may be mentioned the larva of certain flies which are parasitic while the fly itself is not and of the latter *Oxyuris vermicularis* (Linnæus 1767) is a good example.

Animal parasites may be classified into *ectoparasites* or *epizoa* affecting the skin and exterior of the host and *endoparasites* or

differences in the structure and appearance between some of the permanent parasites and their nearest free living affinities. This difference has been brought about mainly by environment.

The permanent parasite particularly if an entozoon has food prepared for it by its host in an easily assimilated form hence there is no necessity for a complicated digestive apparatus which

host will subserve their purpose. Some of them have no necessity as a rule for locomotion for sexual purposes because hermaphroditism is common and even in cases where this is not found parasites often live in couples (male and female). Being enclosed in the body of the host there is no necessity for sense organs therefore these are much reduced or are absent.

On the other hand the parasite requires something to enable it to fix itself firmly to the tissues of the host hence suckers and hooks are often found and also clasping and clinging organs.

The chance of a parasite locked up inside a host successfully reproducing its species is small hence reproduction assumes important features associated with the production of large numbers of new forms which are often protected by shells or other coverings.

Reproduction may be asexual especially in the protozoa or

which the sexual life-history is gone through.

With regard to the sexual process it may be noted that often the male is smaller than the female and in fewer numbers but there are exceptions.

Usually parasites keep strictly to certain hosts called normal

**SECTION C**

**PARASITES**

**ANIMAL PARASITES**

**VEGETAL PARASITES**

metamitosis the polar caps of archoplasm situate in the cytoplasm assist in the mitosis

2 Gemmation—In gemmation the nucleus divides usually by mitosis into two or more nuclei which either travel to the periphery and become surrounded by small masses of protoplasm which separate from the parent parasite—ectogenous gemmation—or remain in the cytoplasm a portion of which becomes differ-

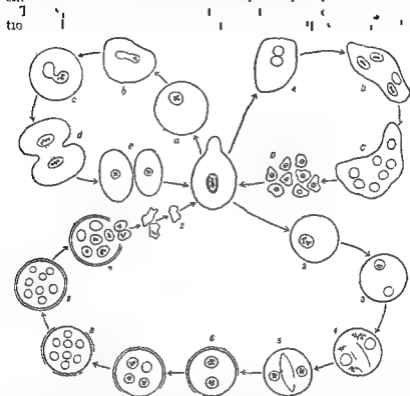


FIG 42 —DIAGRAM OF THE LIFE CYCLES OF *Loeschia coli* LOESCH *emendavi* SCHAUDINN 1903 SHOWING HARTMAN'S AND WHITMORE'S VIEWS AS TO ZYCOSIS

1 *Loeschia coli*: a e stages in the process of binary fission by promitosis A D schizogony by repeated division 2 12 sexual reproduction or sporogony 13 24 are the gametes 25 the zygote but the e stages are not definitely known

hosts but at times they are found in unusual hosts—for example *Echinorhynchus gigas* (Goeze 1782) which is usually found in pigs may infect man. Such a parasite is called a chance parasite while objects mistaken for parasites are known as pseudo parasites.

The above are examples of simple parasitism but there are parasites which are parasitic upon other parasites—a condition called hyperparasitism. These hyperparasites may be secondary, tertiary or quaternary and their importance in disease has been emphasized by Sambon who has shown that it may be one of the causes of the disappearance of malaria from a district for the black spores found by Ross in mosquitoes infected with malaria are now known to be hyperparasites of the genus *Nosema*.

For further remarks see the article on Metazoan Parasites.

**Nomenclature**—Medical men in the tropics are at present almost daily

**1 Language Rule**—The name given to the parasite must be in Latin and

(Loubold 1890) causes a varied number of symptoms which can all be classed together as Paragonimiasis.

The drawback to this nomenclature is that parasites being very often

4417

**Classification**—The animals which cause and convey the diseases of man may be classified into the following subkingdoms. Subkingdom I Protozoa Subkingdom II Metazoa



**Classification**—The classification of the subkingdom Protozoa is by no means satisfactory at present but for remarks upon this subject we refer the reader to Poche's article thereon written in the *Archiv für Protistenkunde* vol 30 in 1913

The old classification was into four classes (1) sarcodina (2) mastigophora (3) sporozoa (4) infusoria

The sarcodina move and capture food by pseudopodia the

more complete knowledge of which is still required

Schaudinn's work with regard to the life-history of flagellates having failed to be confirmed and Hartmann's binucleate theory having fallen into disuse we have returned to Doflein's classification as set forth below

Among the blood parasites of man and other animals there are found some wavy thread like organisms (*Spirochaetes* and *Tre-*  
*p*  
*p*

low type of nucleus but so different are they from the ordinary phyla of protozoa that following Doflein's and Fantham's suggestions we shall place them in a separate section making them an Appendix to the Mastigophora though Calkins thinks it better to leave them in their old position at present

Further the old group of the Sporozoa Leuckart 1879 contains two such different groups of animals in its subdivisions into Telosporidia and Neosporidia that it is justifiable to do away with this combination and to raise the two divisions to the rank of separate phyla

Finally the two nuclei of the infusoria are so totally different in function from the nucleus or nuclei of the rest of the protozoa that the Subkingdom is capable of being divided into two divisions—the Heterokaryota or infusoria and the Plasmodromata under which heading come the other phyla

In the present work the following classification is adopted—

#### DIVISION A PLASMODROMATA DOFLEIN 1901

PHYLUM I—*Sarcodina* Hertwig and Lesser 1874

PHYLUM II—*Mastigophora* Diesing 1866

PHYLUM III—*Telosporidia* Schaudinn 1900

PHYLUM IV—*Neosporidia* Schaudinn 1900

#### DIVISION B HETEROKARYOTA HICKSON 1903

PHYLUM V—*Ciliata* Perty 1852

PHYLUM VI—*Acinetaria* Lankester, 1885

**Plasmotomy**—Plasmotomy is the term applied to the intermediate division of the cytoplasm of multinuclear parasites into two or more masses which afterwards may or may not reproduce by spore-formation

3 **Spore Formation**.—Instead of being considered a process of internal gemmation the formation of pansporoblasts may be looked upon as a process of spore formation proceeding while the organism grows as is typically seen in the Neosporidia

The typical asexual spore-formation or schizogony is however met with in the Telosporidia in which the early stages absorb nutriment and increase in size being therefore called trophozoites. When fully grown they form a quiescent body the schizont whose nucleus and cytoplasm divide into a number of small forms called asexual spores or merozoites generally however leaving a little undivided cytoplasm laden with effete matter which is called a *nucleus de reliquat* or rest body



FIG 43—SCHIZOGONY OF *Plasmodium* AS GRASSI AND FELETTI (After Schaudinn)

1 Young trophozoite 2 ring form 3 ring form showing hemoglobin digestion 4 ring form with highly convoluted nucleus and many small dark granules

These merozoites are the forms by which the parasite multiplies in the given host and are not the means by which new hosts are infected. They therefore enter new cells in the host in which they are formed and growing into a trophozoite complete an asexual life-cycle which is called the cycle of schizogony or simply schizogony (Fig. 43)

A time arrives in the infection of every host when the food material for the given parasite is diminished by the numbers of forms produced by asexual reproduction or when the tissues of the host react against the parasite by chemical substances or phagocytosis or by both methods combined

When these adverse circumstances become sufficiently severe changes take place in the parasite which produce form capable of leaving the given host and existing outside it either in a different species of animal or simply in the exterior until an entry is made

nomenclature was reversed by Schaudinn (as Dobell has pointed out) in 1903 and it is most difficult to alter it at the present time.

**History**—In 1859 Lambl published a note referring to the presence of an amœba associated with other protozoa in the motions of a case of dysentery and this was followed by an account

while Calandruccio infected himself *per os* with the cysts. They both noted its non pathogenicity. Lewis and Cunningham in 1881 saw them in the motions of persons suffering from cholera. In 1894 Celli and Fiocca described and named six species of amœba occurring in man but it is doubtful what these really are probably some of them belong to *L. coli*. Grassi's findings were confirmed by Koch and Kartulis but Kruse and Pasquale in a classical investigation demonstrated that there were two kinds of amœbæ one harmless and the other the cause of dysentery. It was not however until Casagrandi and Barbagallo investigated and defined *L. coli* which is harmless that it was possible for Jürgens to make his researches which extended by Schaudinn ended in defining a second amœba named *L. histolytica* which was considered to be the cause of amœbic dysentery. It has been studied in detail in 1912 by Hartmann and Whitmore with important results and in an important memoir by James in 1914.

It seems to us that Wenyon's researches into *L. muris* are so important with regard to the opposing opinions of Schaudinn and Werner on the one hand and Hartmann and Whitmore on the other that we give this in detail.

#### *Loeselia muris* Grassi 1881

The life history of this amœba has been carefully studied by Wenyon in

nucleus found in *L. coli*

**Species.**—Before commencing the systematic description of the Protozoa it may perhaps be as well to remind the reader that in the higher animals the distinctness of a species depends upon the fertility of its members *inter se* but not usually with members of other species

In protozoology and bacteriology many mere varieties have been called species but such variants *lack any morphologically specific character* and are merely separated from one another by *physiological characters* which as we have seen in the section on evolution of disease can be made to alter by change of environment

It is convenient and useful for purposes of identification and reference to give them names as though they were true species and this does no harm so long as the reader does not expect to find morphologically specific differences in these forms which can only be separated biologically and physiologically

### DIVISION A PLASMODROMATA DOGLEIN 1901

**Synonym** —*Cytomorpha* Hatschek 1888

**Definition** —Protozoa in which the nucleus is not separated into reproductive (micronucleus) and non reproductive (macronucleus) parts

**Classification** —The Plasmodromata may be divided into phyla according to the following scheme

(a) "

(b)

the trophic phase—*Teleosporidia*

II Spore formation and trophic stage proceed simultaneously—*Neosporidia*

er

### PHYLUM SARCODINA Hertwig and Lesser 1874

**Definition.**—Plasmodromata which move and capture their food by means of pseudopodia

**Classification.**—The Sarcodina may be classified as follows —

(a) Without axial filaments in the lobose filose or reticulate pseudopodia—*Rhizopoda*

(b) With central axial filaments in fine ray like pseudopodia—*Heliozoa* Haeckel 1866 *Radiolaria* Haeckel 1861

**Remarks.**—Only the Rhizopoda concern us at present

### CLASS RHIZOPODA DOG SIEBOLD 1845

**Definition.**—Sarcodina parasitic or free living without axial filaments in their lobose filose or reticulate pseudopodia

In 1917 Wenyon and O Connor published exceedingly valuable researches on the diagnosis of *L. coli* and on the house fly as a carrier of the cysts

We therefore see that while a great deal is known as to the structure life-history and method of infection there is still much to be observed. Are Schaudinn and Wenyon correct in their description of autogamy or does *L. coli* really form gametes like *Entamoeba blatta*? Is there any true schizogony?

After this rather long history we will not enter fully into the structure of *L. coli* but will only present a very condensed account

**Morphology**—In diameter it measures as a rule 20 to 40  $\mu$  although forms as small as 10  $\mu$  and even 5  $\mu$  have been described. The cytoplasm is vacuolated and contains bacteria and extremely rarely one red corpuscle. The ectoplasm is not visible until a pseudopodium is about to be protruded. The vesicular nucleus resembles that of the tetragenia stage of *L. histolytica* an account of which is given below but the cyclic changes are not so well defined. The karyosome when present is small and is composed of two chromidia united by a chromatic substance. At the commencement of a cycle this karyosome is a round compact mass of chromatin connected with the periphery by a linen network with few chromatinic granules. This karyosome breaks up its chromatin increasing in amount the linen network becomes thicker and there are more chromatin granules at its nodes and this goes on until all the chromatin is collected as blocks under the nuclear membrane and only a small granule is left in the centre and then the cycle begins again by the growth of the granule into a large karyosome.

**Life-History**—The life cycle comprises two phases a binary division by promitosis takes place (and not by amitosis as described by Schaudinn). The process of schizogony is described to take place by repeated division into two forming two four and eight nuclei (*vide* Fig. 42 p. 292) which form eight little Loeschiae which begin the asexual cycle again but this is very doubtful. Encystment takes place followed by division of the nucleus into two four and finally eight nuclei. During this stage a large vacuole exists in the cytoplasm which disappears in the eight nuclei stage. Contrary to Schaudinn Hartmann and Whitmore believe that no zygosis takes place in the cyst but that eight little amœbæ are formed which on escaping from the cyst they think may conjugate in pairs and form the synkaryon or zygote from which the vegetative forms arise after the manner described by Mercier in 1909 for *E. blatta*. Sometimes the cysts have more than eight nuclei—e.g. ten or twelve—which must be considered to be abnormal.

**Cystic Stage.**—A cyst is spherical or slightly oval, 12 to 14 microns in dia-



FIG 49—DIAGRAM OF THE LIFE CYCLES OF *L. muris* GRASSI (CONSTRUCTED FROM WEYBON'S DRAWINGS)

Each pair then fused to form one nucleus and then almost immediately divided to form four nuclei, and these again to form eight nuclei. During this process the soft gelatinous cyst wall of the precystic stage is converted into a hard, chitinous wall, which an inner membrane

perate Zone. In the former it is especially common in the faces of natives.

**Pathogenicity.**—*L. coli* is a non pathogenic commensal found in man, and possibly in rats and mice, and perhaps in other animals.

### *Loeschia histolytica* Schaudinn, 1903.

**Synonyms.**—*Amœba coli* Loesch, 1875, *Entamœba histolytica* Schaudinn, 1903, *Entamœba tetragena* Viereck, 1907, *L. africana* Hartmann and Prowazek, 1907, *E. minuta* Elmassian, 1909, and *Amœba dysentericæ* Councilman and Lafleur, 1891. Among the many doubtful species of Amœbæ found in man there must be

**Nomenclature.**—The correct name is *Loeschia coli* (Loesch, 1875), as will be explained below.

**Definition.**—*Loeschia* with cytoplasm often containing red corpuscles and with four nucleate cysts.

**Historical.**—*Early Observations.*—Loesch discovered the amœba which he named *Amœba coli* in motions from a case of dysentery, and considered it to be pathogenic. It was by this name that Quinke and Roos in 1893 called the organisms found by them in dysentery. As already stated, Councilman and Lafleur called an amœba found in cases of dysentery *Amœba dysentericæ*, and it is certain that this was Loesch's *Amœba coli*. An amœba somewhat similar to the 'tetragena stage' of *L. histolytica* was seen by Kruse and Pasquale in 1893.

We thus see that the correct name for the dysenteric amœba is *Loeschia coli*, as Dobell has pointed out, and that the correct name for the harmless amœba is *L. hominis* because Casagrandi

in 1893 as there is no  
in this nomenclature  
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is not

**Bionomics**—*L. coli* lives in the lumen of the intestine and feeds upon the contents of the bowel

**Diagnosis**—Amœbæ moving slowly with pale non refractile pseudopodia with a thin rim of ectoplasm and with an endoplasm containing all sorts of organisms and no red cells, and with a large distinct nucleus are *L. coli* and this diagnosis should be confirmed by the discovery of the cyst because—

1 The typical characters are liable to considerable changes and the amœba may be very like *L. histolytica* as will be emphasized below

2 Wenyon and O Connor's researches confirm that *L. coli* will not as a rule at all events ingest red blood-corpuscles

With regard to the

they are to be distinguished by the number of nuclei which in the vegetative form is generally eight



FIG 50—*Loeschia coli* SCHAUDINN VEGETATIVE FORM STAINED  
(X 10,000 DIAMETERS)  
(After James)

**Carriage by House-Flies.**—Wenyon and O Connor working in Egypt have shown that *L. coli* and *L. histolytica* cysts can be ingested by house-flies (*Musca Fannia Calliphora* and *Lucilia*) which have fed on faeces containing such cysts. These cysts can live in the gut of the fly so long as any faecal matter remains there but die after all the faecal matter has been expelled which takes place some twenty four hours after the faecal feed. The cysts may be seen in the droppings of the fly as early as five minutes and as late as twenty four hours after the faecal feed.

As to the conveyance of cysts on the exterior of the fly the observations of Kuenen and Swellengrebel and Nicol show that flies do not move far until they have cleaned themselves so that but little faecal matter is left and as this dries the cysts perish.

**Method of Infection.**—The experiments of Calandruccio and of Wenyon and O Connor have proved that infection takes place for us and is due to the cysts and that further these are introduced most probably by food contaminated by the cyst laden faecal matter of house flies.

**Distribution.**—*L. coli* is found in both the tropics and the Tem



attack of dysentery the chromatin in and around the nucleus becomes  
 and the  
 The  
 These  
 which

spores

*Recent Work*—Modern researches tend to confirm Schaudinn's morphological description as well as his binary fission but not the amitotic form of division while the bud formation (Fig 52) has been proved by James and others to be artificial and the spores are regarded as not belonging to an Amœba but to some other organism in the fœces

In 1905 Craig in the Philippine Islands confirmed Schaudinn's morphological characters and called attention to the rapid movements of *L. histolytica* and to the greenish tint which it often assumes in motions containing much blood

In 1907 Viereck stated that there were more than one pathogenic amœba in man. The second one which he named *Ent. amœba tetragena* looked like *L. coli* but had only four nucleate cysts and this he thinks is the type seen by Quinke and Roos and by Kruse and Pasquale. In the same year Hartmann and von Prowazek found an entamœba in patients coming from Africa which could be differentiated

by its nuclear structure from *L. coli* and *L. histolytica* and this they named *E. africana* but later Hartmann finding quadri-nucleate cysts concluded that it was the same as *E. tetragena* which is an accepted fact

In 1908 Craig drawing attention to variations in *L. histolytica* and in *L. coli* emphasized the difficulty in differentiation between



FIG 52—ARTIFICIAL BUDDING IN *Loeschia histolytica* SCHAUDINN IN MOIST CHAMBER PREPARATIONS

(After James)

same year Noc wrote a paper mainly of an epidemiological nature but also dealing with this amœba and Elmassian described

clearly visible unless coloured by some preparation. When resting it is oval or spherical but during movement it alters its appearance repeatedly throwing out pseudopodia and creeping about. The nucleus is small about  $5 \mu$  in

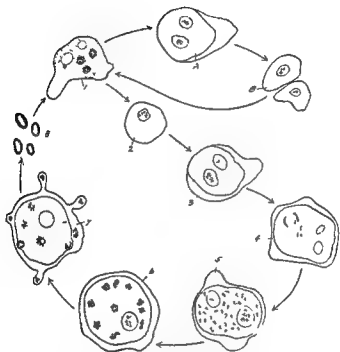


FIG 51—DIAGRAM OF THE LIFE CYCLES OF *Loeschia histolytica* ACCORDING TO SCHAUDINN'S VIEW<sup>s</sup>

(Constructed from Craig's drawings in the *Journal of Infectious Diseases*)

A B Binary fission 1-8 spore formation

1 Binary Fission—This method was only discovered by Schaudinn on examining fresh specimens of the infected alimentary canal

The nucleus divides by amitosis into two and then the cytoplasm splits into two equal daughter cells

2 Gemmit -

3 Spore Formation—In spore-formation, which only takes place under favourable circumstances such as when recovery is taking place after an



*E. minuta* which is to day generally considered to be a stage of *L. histolytica*

In 1911 there appeared a valuable paper by Walker, in which he distinguished only *L. coli* and *L. histolytica* but the latter was considered to have 'a tetragena stage' a fact accepted to-day

perceptible layer on the inner surface of the nuclear membrane,

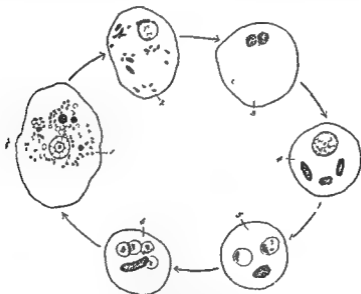


FIG. 53.—DIAGRAM OF THE LIFE CYCLES OF *Loeschia histolytica* SCHAUDIN  
(Constructed from Hartmann's drawings in the *Archiv f. r. Schiff's und Tropen Hygiene*)

1 Fully grown parasite 2 6 stages in encystment and nuclear division

with or without a few fragments scattered in the nuclear network (*histolytica* stage) or as a more extensive but loose peripheral granular layer and a loose central larvosome (*tetragena* stage)

red blood cells but at times these are wanting. It may be vacuolated.

It possesses a very delicate membrane, with a few peripherally arranged grains of chromatin.

In stained specimens there is rarely any differentiation of the ectoplasm from the endoplasm. The cytoplasm may contain vacuoles erythrocytes and perhaps the phagocytosed nuclei of other cells. The nucleus unless distorted, is roundish, possesses a delicate nuclear membrane, under which a thin band or a few grains of chromatin may lie. The centre of the nucleus is occupied by a

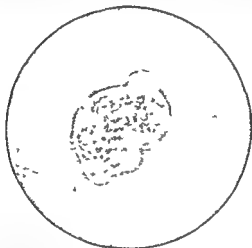


FIG. 54.—PHOTOMICROGRAPH OF THE LIVING AND RAPIDLY MOVING *Loeschia histolytica* SCHAUDINN, IN THE YOUNG TROPHOZOITE STAGE OF ACUTE DYSENTERY ( $\times 1500$  DIAMETERS)

(From the *Journal of Tropical Medicine*)

karyosome, which may contain a very minute centriole. The space

distinguish it from the true chromatin.

*Old Trophozoite Phase*—This is the phase so carefully described

In the same year there appeared papers by Wenyon and O'Connor on the Human Intestinal Protozoa in the Near East detailing the characters of *L. histolytica* and by Chalmers and O'Gravall with regard to its presence in the urinary tract which brings in the synonym *Amoeba urogenitalis* Baetz 1883

Wenyon and O'Connor consider that no infection can be ascribed to *L. histolytica* unless some amœbæ with included red corpuscles are present or unless typical cysts are present in the stool. An

- 1 Amœbæ containing red blood corpuscles are *L. histolytica* whether the stool is dysenteric or not and further they indicate an active dysentery. These cases urgently require emetine treatment.
- 2 Amœbæ none of which contain red blood corpuscles occurring in a dysenteric motion are indicative of *L. coli* or of *L. histolytica* in a carrier while the cause of the dysentery is not an amœba and these cases should be watched for a few days without treatment with a view to finding the cysts.
- 3 Amœbæ none of which contain red blood corpuscles occurring in non-dysenteric motions may be *L. coli* or *L. histolytica* and diagnosis has to be made by finding the quadrinucleate cysts perhaps after several days observation.

In 1918 Dobell showed by experimental infection of tadpoles that *I. histolytica* and *L. ranarum* were distinct species and with Jepps drew attention to the existence of diverse races of *I. histolytica* which could be distinguished by the dimensions of the cyst.

The above is a summary of the work of the author and his colleagues in the study of the life history of the amoeba. It is published in the *Journal of the Royal Microscopical Society*, 1918, 37, 115.

It occurs when active dysenteric processes are proceeding in the bowel and is represented as a rule in the fresh condition by large amœbæ measuring from 30-90 microns in diameter but exceptionally being present in small size. As a rule its motility is very marked often starting with such a rapid action as to be worthy of the name explosive. The pseudopodia are broad and may be solely ectoplasmic or be composed of endoplasm as well. The ectoplasm may be clearly distinguishable from the endoplasm even when the amœba is at rest but often there is no such distinction.

The cytoplasm may have a well-defined light green colour or more usually this colour is wanting. It may contain a number of vacuoles or it may not. It often possesses a number of ingested

**Loeschia williamsi** Prowazek 1911

This *Loeschia* is considered to be identical with *L. coli* Loesch

**Loeschia brasiliensis** H. Baurepauze Aragao 1917

Resembles *L. coli*. Cysts 7 to 15  $\mu$  in diameter with eight nuclei and a double contour membrane. The cysts are characterized by the presence of a certain amount of siderophile substance which divides the cysts into two portions of nearly equal size.

**Loeschia butsehlii** Prowazek 1917

**Synonym** — *E. tanaka butsehlii* Prowazek 1912

Found in a boy in the Caroline Islands. It varies in size from 10 to 24  $\mu$ . Coarse alveolar cytoplasm, nucleus vesicular, round karyosome and centriole. Cysts roundish, said to differ from those of *L. coli*.

**Loeschia mortinatalium** Smith and Weidman 1910

**Synonym** — *Entamoeba mortinatalium* Smith and Weidman 1910 and perhaps *Amoeba pulmonalis* Artault 1898

**Definition** — *Loeschia* of large size, 22-38  $\times$  20-25 microns, with nucleus 10 microns in diameter, with well defined membrane, large karyosome and occasionally a centriole.

**Remarks** — Somewhere about 1890 Ribbert found amœbæ in the kidneys and parotid glands of infants. In 1898 Artault observed amœbæ with a nucleus and a vacuole in a lung cavity. Brumpt has seen similar amœbæ and R. Blanchard has found some in the lungs of sheep which may or may not be the same as the *Entamoeba* or *is* Swellengrebel 1914 found in the gut of sheep. This latter measures 12-14  $\times$  11-12 microns. Its cysts are 8 microns in diameter, uninucleate with a glycogen vacuole. In 1904 Jesionek

sputum and in the lungs of a case of pneumonia in the Anglo  
 many known history of dysentery  
*L. histolytica*. Time must show

**Loeschia minutissima** Brug 1917

**Synonym** — *E. minutissima* Brug 1917

A very small amœba, 4-11  $\times$  4-8 microns. Usually 6-5-7  $\times$  5-6 microns.

**Loeschia tenuis** Kuenen and Swellengrebel 1917

This amœba which was described as *Entamoeba tenuis* measures 6-9 microns in diameter, with cysts 6-8 microns and one to four nucleated, is very like *L. ana* of Wenyon and O'Connor and the *L. minuta* of Woodcock and Penfold which latter however is said to be the same as *E. histolytica*.

by Hartmann under the heading *L. tetragena*, and often called the *tetragena phase*. It resembles the *histolytica phase* in many par-

less zone between which and the nuclear membrane there is a thin network on which granules of chromatin are distributed

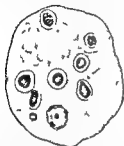


FIG 55—*Loeschia histolytica* SCHALDINN, YOUNG TROPHOZOITE FIXED AND STAINED SHOWING NUCLEUS AND PHAGOCYTED RED BLOOD CELLS ( $\times 1950$  DIAMETERS)



FIG 56—*Loeschia histolytica* SCHALDINN, OLD TROPHOZOITE OR TETRAGENA PHASE STAINED PREPARATION ( $\times 1950$  DIAMETERS)

(After James)

This nucleus undergoes cyclical changes but in a given preparation as a rule, all the amoebae show the same appearance. The cyclical changes are —

1. Large blocks of chromatin under the nuclear membrane, a very



FIG 57—NUCLEUS OF *Loeschia histolytica* SCHALDINN, OLD TROPHOZOITE PHASE STAINED PREPARATIONS ( $\times 1950$  DIAMETERS)

(After James)

Note the cyclical changes described in the text

3. Chromatin more concentrated under the nuclear membrane and at the karyosome margin while a clear zone is appearing around the centriole (Fig. 57, c)

4. Chromatin under the membrane and at the karyosome margin (Fig. 57, d)



**Vahlkamfla lobospinosa** Craig 1912

**Synonym** — *Amœba lobospinosa* Craig 1912 *V withmorei* Hartmann 1912

This amœba was cultivated from a dysenteric stool from a patient in Manila in 1905 and first described by Musgrave and Clegg. In 1912 it was studied by Craig and called *Amœba lobospinosa* also by Williams and Calkins by James by Liston and by Wells.

James in the Canal Zone obtained this amœba for a considerable period from the fœces of a patient which were guarded against contamination and hence it must be admitted that it can live for a time in the intestine of man but it is probably non pathogenic.

On the other hand in all the other reported cases it has occurred as an aerial contamination of the fœces or pus in which it has been found. It was first cultivated by Musgrave and Clegg.

## OTHER SPECIES

es of *Limax*  
the *Pseudo*  
! *Vahlkamf*

1904 as the type species and allowing that *V lacustris* Naegler 1900 is the same species the following are known

A *Small forms* 3-15 microns —

Cysts 1-5 microns in diameter—*V limax* Dujardin 1841 emendavit Vahlkamf 1904

Cysts over 7 microns in diameter

(a) Karyosome surrounded by a peripheral row of chromatin granules—*V lacerta* Hartmann 1907

(b) Karyosome not so surrounded—*V froschi* Hartmann and Prowazek 1907

B *Medium sized forms* 15-30 microns —

(a) Trophozoite binucleate—*V diploidea* Hartmann and Naegler 1908

(b) Trophozoite uninucleate —

1 Contractile vacuole present—*V tachypoda* Glaeser 1912

2 Contractile vacuole absent—*V poly-poda* Schutze 1875

C *Large forms reaching to 50 microns* —

(a) Trophozoite binucleate—*V binucleata* Gruber 1884

(b) Trophozoite uninucleate —

1 Ectoplasm like a lamella—*V lamellipoda* Glaeser 1912

2 Ectoplasm not so distinct —

(1) Nuclear division promitotic—*V albida* Naegler 1909

(2) Nuclear division mesomitotic—*V guttula* Dujardin 1912

*Loeschia* in Animals—Though somewhat beyond the bounds of the present work we may mention that amoebæ believed to belong to the genus *Loeschia* but requiring restudy in the light of recent researches occur in several vertebrates—e.g. *L. nitida* Castellani, 1908 found in liver abscesses and dysentery in monkeys in Ceylon *L. cobaya* in guinea pigs *L. enterica* in cats etc *L. muris* Grassi in mice considered by Wenyon to be *L. coli* *L. fecalis* in several animals *L. intestinalis* in horses etc *L. gallopavæ* in turkeys *L. ranarum* Grassi in frogs and many more



FIG 61—*Loeschia nitida* CASTELLANI 1908 CONTAINING RED BLOOD CELLS

**Genus *Vahlkamfia* Chitton and Lalung Bonnaire 1912**

**Definition**—*Gymnamoebida* with vesicular nucleus (protozoaryon) having one large karyosome with or without a centrosome with little peripheral chromatin with division by promitosis. Cysts typically uninuclear

**Remarks**—Practically all cultivable amoebæ isolated from human stools and potable water by various observers belong to this genus. Cropper has grown a peculiar amoeba belonging to this genus in citrate solution.

**Type**—*Vahlkamfia limax* Dujardin 1841 *emendavit* Vahlkamfi 1904

***Vahlkamfia punctata* Dangeard 1910**

This amoeba was found in Indo China in the motions of a case of diarrhoea. The ectoplasm is rarely visible and the endoplasm is



FIG 62—AMOEBÆ AND CYST FOUND IN HUMAN FECES AND POSSESSING THE LIMAX TYPE OF NUCLEUS (a) TROPHOZOITE (b) CYST (X 1950 DIAMETERS)

(After James from the *Annals of Tropical Medicine and Parasitology*)

very ba  
cysts are  
contour

The  
double  
te

*Vahlkamfla lobospinosa* Craig 1912

Synonym.—*Amœba lobospinosa* Craig 1912 *V withmoresi* Hartmann 1912

James in the Canal Zone obtained this amoeba for a considerable period from the fæces of a patient which were guarded against contamination and hence it must be admitted that it can live for a time in the intestine of man but it is probably non pathogenic

On the other hand in all the other reported cases it has occurred as an aerial contamination of the fæces or pus in which it has been found It was first cultivated by Musgrave and Clegg

## OTHER SPECIES

In 1917  
amoeba intc  
lsmar but  
1904 as tr  
1900 is the same species the following are known

## A Small forms 3-15 microns —

Cysts 15 microns in diameter—*V lsmar* Dujardin 1847  
*emendavit* Vahlkamf 1904

Cysts over 7 microns in diameter

- (a) Karyosome surrounded by a peripheral row of chromatin granules—*V lacerta* Hartmann 1907  
(b) Karyosome not so surrounded—*V froschi* Hartmann and Prowazek 1907

## B Medium sized forms 15-30 microns —

(i) Trophozoite binucleate—*V diploidea* Hartmann and Naegler 1908

(b) Trophozoite uninucleate —

- 1 Contractile vacuole present—*V tachypodia* Glaeser 1912  
2 Contractile vacuole absent—*V polypodia* Schutze 1875

## C Large forms reaching to 50 microns —

(a) Trophozoite binucleate—*V binucleata* Gruber 1884

(b) Trophozoite uninucleate —

- 1 Ectoplasm like a lamella—*V lamellipodia* Glaeser 1912  
2 Ectoplasm not so distinct —  
(1) Nuclear division promitotic—*V albidus* Naegler 1909  
(2) Nuclear division mesomitotic—*V guttula* Dujardin 1912

eggs and embryos of a crab belonging to the genus *Peltogaster*

*Vahlkamfia nana* Wenyon and O'Connor 1917

Synonym—*Entamoeba nana* Wenyon and O'Connor 1917

*Vahlkamfia nana* Brug 1917

Definition.—*Vahlkamfia* of small size (5 to 8 microns) moving slowly with blunt ectoplasmic pseudopodia nucleus with membrane and large central karyosome cysts  $7.8 \times 8.10$  (when elongated) microns with one to four nuclei without chromidial bodies



Remarks—This amoeba was found by Wenyon and O'Connor in Egypt and we have seen it in the Anglo Egyptian Sudan and Southern Italy. It has been confused with *L. culi*, *L. histolytica* and *V. limax*. *V. nana* has not been cultivated.

Time will be required to show definitely its generic position. Provisionally we have placed it under *Vahlkamfia* though its cyst is binucleate.

Genus *Dientamoeba* Jepps and Dobell 1918

Definition—Cyst with two nuclei without a known flagellum both nuclei of the same size

One species.—*Dientamoeba fragilis* Jepps and Dobell 1918 found in man. It is the only known species at present.

*Dientamoeba fragilis* Jepps and Dobell 1918

Definition.—*Dientamoeba* with the generic characters

Historical.—This amoeba was first detected by Jepps and Dobell in 1917 in a native of the British Isles who had never been abroad

but was suffering from slight diarrhoea attributed to a chill. It was found in British soldiers who had been to Salonika and in natives of New Zealand serving as soldiers. In all it has been seen in seven cases.

**Morphology**—It is a rounded some 3.5-8.10 by means of extremely of sharply defined ectoplasm. The rest of the body is often rounded and consists of granular endoplasm and is situated posterior to the pseudopodia thus giving a snail-like appearance during active movement.

The cytoplasm is alveolar and contains bacilli and cocci. There is no contractile vacuole but there are diffuse brown stained patches indicative of glycogen in iodine stained preparations. The amoeba is binucleate in about 80 per cent of the forms examined and these nuclei are usually invisible in the living organism. In stained preparations they are 2 microns in diameter and each contains a chromatin granule on which there is no chromatin. There is sometimes a separate granule to be seen lying in the centre of the karyosome which is the centriole of many authors.

**Life-History**—No signs of division or cyst formation have been observed.

**Habitat**—The intestine of man probably in the colon contents

**Binucleate Amoebæ**—We have already noted under the genus *Vahlkamfia* two binucleate amoebæ in addition to *V. ana*—viz *V. diploidea* Hartmann and Naegler 1908 with occasional uninucleate forms and *V. binucleata* Gruber 1884 and have shown that they probably are not *Vahlkamfia* and equally they are not *Dientamoeba*. Another binucleate form may be *Amoeba infra* Glaeser 1912 about which there appears to be much doubt as to whether the name was given to a binucleate or uninucleate form. Schaudinn's *Paramoeba* is a marine binucleate amoeba and forms a genus in which parasitic amoebæ are dissimilar one being found in *Craigia* (*v. de infra*) may also possess two dissimilar bodies one nucleus and the other like a Nebenkoerper.

#### Genus *Craigia* Callans 1912

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body

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called *Nehelkamas* D.

In 1913 Barlow discovered a new species which he called *Craigia migrans* in Honduras.

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*Craigia migrans*

*Craigia hominis* Craig 1916

Synonym — *Pyrenocystis*

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*Craigia migrans* Barlow 1913

Definition.—*Craigia* in which the accessory nuclear body is absent and in which the swarm spores do not divide longitudinally before becoming amoebae.

History.—It was discovered by Barlow in fifty-one infections in Honduras.

**Morphology**—*O. hominis* is a round or pear shaped parasite averaging from 8 to 10  $\mu$  in diameter with a long flagellum projecting from the more pointed end. There is no undulating membrane. The nucleus is small indistinct and usually situated near the flagellar extremity.



FIG. 68—*Oicomonas hominis* (DAVAINE)

1, 3 and 5 Flagellate forms  
4 encysted form

**Life-History**—Reproduction is by binary fission and Perroncito and P. L. Cardi have described encystment.

**Habitat**—According to our experience at autopsies the parasite may live not only in the small intestine as generally stated but also in some cases in the large intestine. *Oicomonas* is readily killed as observed by Castellani and Willey by a solution of methylene blue (1 in 3 000).

#### *Oicomonas vaginalis* Castellani and Chalmers 1909

**Synonym**—*Cercomonas vaginalis* Castellani and Chalmers 1909  
12  $\mu$  in small  
It is

#### *Oicomonas perryi* Castellani 1907

**Synonym**—*Circomonas perryi* Castellani 1907

but cysts can be seen

**Other Species**—*O. anatis* Davaigne in the alimentary canal of ducks  
*O. canis* Gruby and Delafond in dogs  
*O. gallinarum* Davaigne in fowls

#### Bodonidae Butschli 1884 emendatist Doflein 1901

**Definition**—Monozoa free living or parasitic with one anterior and one posterior (or trailing) flagellum with or without a kinetosome.

5 family represent the  
Dujardin 1841 is in

**Classification**—The following is a poor attempt to differentiate the genera of the Bodonidae known to us—

A With an undulating membrane—

I Kinetonucleus well marked—*Trypanoplasma*

II Kinetonucleus poorly marked—*Trypanophis*

II Without an undulating membrane but a kinetonucleus may or may not be present --

I While swimming all flagella are posterior (Genera with which we are not concerned as yet not found in man)

II "

1 Food believed to enter anteriorly --

(1) Kinetonucleus absent—*Bodo*

(2) Kinetonucleus present—*Procaezekia*

2 Food believed not to enter anteriorly (Genera with which we are not concerned as not yet found in man)

*Trypanoplasma* Laveran and Mesnil 1901 *emendavit* 1904

Parasite

"

undulating membrane

**Life-History** -- Division is longitudinal the kinetonucleus dividing first and then the flagella

Often *Trypanoplasmatata* show seasonable variation the infection being more intense in hot weather. They are evidently pathogens producing animals associated with serous fluid in the peritoneum, pleuridium and uddia of the organs

The parasite appears to be spread by leeches in which the sexual forms conjugate by a fusion of nuclei after reduction and of the cytoplasm from which results an ookinete possessing a trophonucleus and a kinetonucleus

These ookinetes give rise to male and indifferent forms which

Another genus of leech which spreads these parasites is *Hem*

**Classification** -- Crawley considers that the generic name should be *Cryptob* Lecky but the diagnosis of this genus is vague. With regard to classification



Keysseltz is of the opinion that all so far described species should be considered to belong to one species—*Trypanoplasma borreli* L. and M.

Type Species —*Trypanoplasma borreli* Laveran and Mesnil 1901

*Trypanoplasma borreli* Laveran and Mesnil 1901

In the blood of *Leuciscus erythrophthalmus* (the rudd) and *Phoxinus phoxinus* (the minnow) and in the alimentary canal of the leech *Piscicola geometra* and perhaps in *Hirudo medicinalis*

*Trypanoplasma cyprini* Plehn 1903

In *Cyprinus carpio*

*Trypanoplasma (Cryptobia) dendrocoeli* Gantham and Porter 1910

This parasite measured 20 to 40  $\mu$  in length with a large and often curved kinetonucleus. It lives in the alimentary canal of *Dendrocaelum lacteum* and was the first trypanoplasma to be found in the Platyhelminthes

*Trypanoplasma intestinalis* Léger 1905

#### *Trichomonas*

These he considers to be female forms and says that he has seen conjugation with male forms

*Trypanoplasma ventriculi* Keysseltz 1906

Synonyms —*Heteromita dahlis apstena* = *Diplomastix dahlis*

This is found in the intestine of *Cyclopterus lumpus* and is apparently a typical trypanoplasma. Discovered by Dahl in 1887

*Trypanoplasma variem* Léger 1904

In *Cobitis barbatula* (loach) and in *Hemicleptis marginata*

*Trypanoplasma guernei* Brumpt 1905

In *Cottus globio* and develops in *Piscicola*

*Trypanoplasma barbi* Brumpt 1905

In *Barbus fluviatilis* and in the leech (*Piscicola*)

*Trypanoplasma abramidis* Brumpt 1905

In the bream (*Abramis drama*) and the leech (*Hemicleptis*)

*Trypanoplasma truttæ* Brumpt 1905

In *Salmo fario* and perhaps in *Piscicola*

#### Other Species

*T. Keysseltzi* Minchin 1909 in the tench *T. gurneyorum* Minchin 1909 in the pike *T. clariae* Mathus and Léger 1911 in *Clarias macrocephalus* *T. congru*

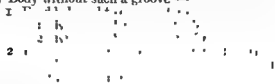
*Trypanophis* Keysseltz 1904

Bodonidæ in *Calenterrata* with two flagella, an anterior and a posterior. The kinetonucleus is situated anteriorly and is much smaller than the trophonucleus. According to Floyd a blepharoplastic granule gives rise to the free flagellum. The attached flagellum arises near the basal granule of the free flagellum and gives rise to a narrow undulating membrane.

*Trypanophis grobbeni* Poche 1903

In the gastrovascular system of different Siphonophora—e.g. *Halistemma ergestinum*. The parasite is curved somewhat like a trypanosome.

- B Without an undulating membrane, but a kinetonucleus may or may not be present —
- I. While swimming all flagella are posterior (Genera with which we are not concerned as yet not found in man)
  - II While swimming all flagella are not posterior —
    - (a) Body with antero-posterior groove—*Colponema*
    - (b) Body without such a groove —



*Trypanoplasma* Laveran and Nicolle 1901 *amendavi* 1904

Definition.—Dodonata living in the blood and alimentary canal of vertebrates

mat  
con  
chr  
con  
run

Often *Trypanoplasma* show seasonal variation the infection being more intense in hot weather. They are evidently pathogenic producing anaemia associated with serous fluid in the peritoneum pericardium and oedema of the organs.

The parasite appears to be spread by liches in which the sexual forms congregate.

Classification.—Crawley considers that the generic name should be *Trypanella* Leidy, but the diagnosis of this genus is vague. With regard to classification,

***Prowazekia urinaria* Hassall 1859**

This species has several times been found in human urine which has been passed some hours

measurement a carrot shaped form varying from 6 by 3  $\mu$  to 25 by 4  $\mu$ . The cytoplasm contains a large number of small highly refractile granules and contains a trophonucleus and a kinetonucleus which

sausage  
a round  
measure-

is a cytostome situate near the root of the short flagellum

**Bionomics**—It moves in a jerky manner with the short flagellum directed forwards and the long flagellum backwards. The small flagellum is also useful in capturing food such as bacteria. Food enters by means of the cytostome and forms the usual food vacuoles which accumulate at the aflagellar end. A contractile vacuole is seen in large flagellates and may measure 1 to 3  $\mu$  in diameter. It is situate near the base of the cytostome to which it is joined by a minute canal. It contracts every 15 to 30 seconds at a temperature of 20° C. It is thought to be the dilated fundus of the cytostome.

**Life-History**—It divides in two by binary fission the blepharoplast apparently dividing first and forming two new flagella after which the cell nuclei and the cell divide. It can lose its flagella and can form round or oval cysts 5 to 7  $\mu$  in diameter inside from which after a time it again becomes flagellate and escapes in its typical form.

**Cultivation**.—In association with bacteria it grows well in urine on salt agar nutrient agar serum agar blood agar peptone salt solution nutrient broth and diluted blood serum at a temperature of 20° C. but is killed by a temperature of 37° C. in one or two hours. It has not been cultivated free from bacteria.

***Prowazekia asiatica* Castellani and Chalmers 1910**

Genus *Prowazekia* Hartmann and Chagny 1910

*Bodo sensu*  
cepted for

example Alexeteff and others oppose it stating that the generic name for all the species included under *Prowazekia* should be *Bodo*, while that for the only species at present under *Bodo*—viz *B. lacerta* Grassi 1881—should be *Prowazekella* (new genus) *lacerta* Grassi 1881. Nor is this the only confusion with regard to *Prowazekia* for one species—*P. urinaria* Hassall 1859—has been found in

contaminations of the urine and faeces after being passed out of the body, and the urine problem is further complicated by the question

forms are harmless occasional parasites of man

Classification. —The species may be differentiated as follows —

A Posterior flagellum free —

1 *Large forms* More than 9 microns in length as a rule —

(a) Shape oval —

1 Rhizoplast present —

(1) Cytostome present—*Urinaria*

(2) Cytostome absent—*Isatisca* and *usquidiss.*

2 Rhizoplast absent—*Cruzii*

(b) Shape pyriform —

Apex sharp—*Weinbergi*

11 *Small forms* Not exceeding 8 microns in length—*Parisi*

B Posterior flagellum attached to the body for a short distance

—*Javanensis*

**Prowazekia cruzi** Hartmann and Chagas 1910

*O. 1400 - 1 - 25 - 1 - 11 0 1 - 1 - 142 - 1 - 1*

believes to be identical with *P. cruzi*

**Prowazekia weinbergi** Mathis and Leger 1910

Pear shaped but rather drawn out into a point length 8 to 15  $\mu$  breadth 4 to 6.5  $\mu$  Flagella at broad end Found frequently in the motions of men in Tonkin

**Prowazekia parva** Naegler 1910

Characterized by its small size the longest forms being 5 to 8  $\mu$  The cysts do not contain flagella

**Prowazekia javanensis** Flu 1912

**Definition**—*Prowazekia* in which the posterior flagellum is attached to the body for a short distance

**Remarks**—Flu believes that there is only one species of *Prowazekia* He obtained his variety from an agar culture of human faeces in the Dutch East Indies

**Prowazekia vaginalis** Castellani and Chalmers 1918

**Definition**—*Prowazekia* living in the vaginal mucus.

**Remarks**—Morphologically identical with *P. asiatica* found in motions but the investigated strains of the latter will not live in vaginal mucus

**Genus Bodo** Stein 1875

**Definition**—Bodonidæ without undulating membrane or kinetoculus but with a rhizoplast While swimming one flagellum is anterior and the other trailing without antero-posterior groove

**Bodo stercoralis** Porter 1918

Discovered in human faeces by Miss Porter Body measures from 14  $\mu$  to 19  $\mu$  long and is from 6  $\mu$  to 9  $\mu$  broad with large nucleus

**Bodo lens** Muller 1,86

**Synonyms**—*Monas lens* *Heteromita lens*

**Remarks**—Usually free living but said to be found in man once (*vide* Animal Parasites of Man by Faubam Stephens and Theobald)

**Genus Toxobodo** Sangiorgi 1917

**Definition**.—Bodonidæ of semilunar shape

**Type and only Genus**.—*Toxobodo intestinalis* Sangiorgi 1917

**Toxobodo intestinalis** Sangiorgi 1917

A flagellate organism semilunar in shape 6-9.6  $\times$  1.6-4.8 microns found in the human intestine and grown for ten generations in culture media (peptone water) It has two flagella and resembles a *Bodo* except in shape

containing food vacuoles but no contractile vacuole. The tropho- nucleus is usually situated in the flagellar third of the cytoplasm and consists of a nuclear membrane and a wide space for the enchyloma, and a central karyosome with usually a centrosome. The kineto- nucleus is situated nearer the flagellar extremity and is connected by a long strand with a small piece of chromatin situated near the aflagellar extremity and by another strand with one of the two blepharoplasts which lie adjacent to the flagellar extremity. These blepharoplasts are united together and as already stated to the kintonucleus by strands. Usually there are two which may lie



FIG. 69.—*Prowazekia asiatica* CASTELLANI AND CHALMERS FROM IRON FACES

takes place by metamitosis but sexual reproduction is unknown. Cyst formation has been observed resulting in rounded bodies 6 to 7  $\mu$  in transverse diameter possessing tropho- and kinto- nuclei and enclosing the remains of the flagella.

**Culture**—*P. asiatica* is readily cultivated in liquid and in the water of condensation of solid media in symbiosis with bacteria. The most suitable medium is the condensation water of nitroso agar (2 to 4 per cent) or maltose agar on which a few drops of



FIG. 70.—*Prowazekia asiatica* CASTELLANI AND CHALMERS FROM CULTURE. (After Whitmore.)

albumin water have been placed when it can be subcultured and grown indefinitely if the tubes are kept uncapped and subcultures are made twice a week.

**Pathogenicity**—Probably nil

*MASTIGOPHORA AND PROTOMONADINA*

nn and Chagas met with it in Brazil. Since then it has only

measuring on an average  
width, but Wenyon has  
-3 microns in greatest



FIG 71 —*Cercomonas longicauda* DUJARDIN, 1841  
Type with granules around the nucleus, compare this with Fig 74  
(Wenyon from the *Quarterly Journal of Microscopical Science*)

The cytoplasm is alveolar and contains a large anteriorly situate nucleus, which has a nuclear membrane enclosing a clear space, in which lies a large karyosome. The nuclear membrane may be drawn out into a cone, at the apex of which lies the granule-blepharoplast—from which either the flagella spring directly or a single rhizoplast passes to the periphery and then divides into the two flagella. The flagellum from the anterior broader end reaches the posterior end when it becomes a free posterior flagellum. The cytoplasm also contains a number of bright refractile granules.



FIG 72 —*Cercomonas longicauda* DUJARDIN, 1841  
Type without granules and Cyst  
(After Wenyon)

**Life-History.**—Simple fission, with division of the nucleus by promitosis, takes place, while cyst formation is also known.

The cysts measure some 6-7 microns in diameter. They are slightly brownish spherical bodies containing a spherical central nucleus surrounded by bright refractile granules.

**Pathogenicity.**—It is believed to be non-pathogenic, and to be accidentally present in the faeces.

Genus *Heteromita* Dujardin, 1841

Bodonidæ round or oval, with two flagella, one at each pole

*Heteromita zeylanica* Castellani and Chalmers 1910

*Heteromita zeylanica* Castellani and Chalmers 1910  
 This species is characterized by its oval shape and two flagella. It is found in ankylostomiasis in Ceylon.

ORDER CERCOMONADIDÆ Saville Kent 1880 *emendavit* Butschli

**Definition.**—Monozoa with elongate or oval forms, possessing one free anterior flagellum and one trailing flagellum

**Type Genus.**—*Cercomonas* Dujardin 1841 *emendavit* Wenyon, 1910

**Remarks.**—It appears to us that this is the only genus which can be classified in this family at the present moment

Genus *Cercomonas* Dujardin 1841, *emendavit* Wenyon, 1910

*Cercomonas* is characterized by its oval shape and two flagella. The flagella are attached to the margin of the cytoplasm, at which it divides into the two flagella, one of which is anterior, while the other is posterior and closely attached to one side of the body at the posterior end of which it becomes free.

**Type Species.**—The type species of the properly defined family is certainly *Cercomonas longicauda* Dujardin 1841 *emendavit* Wenyon 1910, even though the first species in Dujardin's description is *C. zeylanica*.

We consider it to be a synonym

There are a number of species described by Dujardin but until they have been examined by modern methods it is impossible to define them. They are all free-living forms

*Cercomonas longicauda* Dujardin, 1841 *emendavit* Wenyon 1910

**Synonyms.**—*C. zeylanica* Castellani and Chalmers 1910  
*C. zeylanica* Wenyon 1910  
 microns, with very

*C. longicauda* was first discovered by Dujardin in 1841 in an old infusion, and was next described by Wenyon in 1910, being found in cultures made from human faeces. In the same year



**Genus *Enteromonas* da Fonseca 1915**

**Definition** — *Embadomonadinae* without a cytostome or trailing flagellum and with three anterior flagella

**Type and only Species.** — *Enteromonas hominis* da Fonseca found in Brazil

***Enteromonas hominis* da Fonseca 1915**

**Definition** —

**Remarks** — Found in 1915 and

Anglo Egyptian Sudan in Europeans and natives

**Morphology** — The parasite is roundish or oval without a tail and with a diameter varying from 5-6 microns. The periplast is not rigid and encloses an endoplasm often with inclusions such as bacteria. Situate anteriorly lies the protokaryon type of nucleus from which a rhizoplast runs to a blepharoplast from which three anterior flagella arise.

**Life-History** — Da Fonseca records longitudinal division.

**Pathogenicity** — The flagellate probably causes diarrhoea.



FIG 73 — *Enteromonas hominis* DA FONSECA 1915

**Genus *Embadomonas* Mackinnon 1911**

**Synonym** — *Waskia* Wenyon and O Connor 1916

**Definition** — *Embadomonadinae* with a cytostome and one anterior and one posterior flagellum and with a siderophilous often folded cytostomic margin.

**Type Species** — *Embadomonas agilis* Mackinnon 1911

**Other Species** — The type and the other species may be recognized as follows —

(a) *Habitat* intestine of *Trichopterus* and *Typhlops* British Isle borders fecus cytostome excised and



74 — *Embadomonas agilis* MACKINNON 1911

Mackinnon's Journal (1911)

3

about 4

mirrored  
c 1

*Cercomonas parva* Hartmann and Chagns 1910

This is probably the same as *C. longiculus*

FAMILY TETRAMITIDÆ Kent 1880 *emenda* et Chalmers and Pekkola, 1917

**Definition**—  
 exception of E  
 one anterior ar  
 or without a pc  
 form an undul  
 cy<sup>6</sup> — " "

as follows —

A *Without an avostyle* —

(a) With three flagella—Subfamily I *Embodomonadinae*

(b)

B *With an avostyle*—Subfamily III *Trichomonadinae* Chalmers and Pekkola 1917

## SUBFAMILY EMBADOMONADINÆ Chalmers and Pekkola 1918

**Definition**.—Tetramitidæ with or without a cytostome but without an avostyle and with three flagella only (Only two are visible in Embodomonads)

**Classification**.—The known genera of the subfamily Embodomonadinae may be recognized as follows

A *Without cytostome* —

I With three anterior flagella—(1) *Enteromonas* de Fonseca 1915

II With one anterior and two posterior flagella—(2) *Dillengeria* Saville Kent 1880

III With two anterior and one posterior flagella—(3) *Dicercomonas* Chalmers and Pekkola 1919

B *Cytostome present or probably present (as a groove) —*

I With one anterior one cytostome and then free and one free trilineal flagellum (4) *Trimastix* Saville Kent 1880

II With one anterior and one posterior flagellum which is generally cytostome and with a large cytostome with siderophilous often folded border—(5) *Embadomonas* Wickham 1911

The genera in which we are interested are *Enteromonas*, *Dicercomonas* and *Embadomonas*

- Tetramitus* Perty 1852  
*Callodictyon* Carter 1865  
*Costiopsis* Senn 1900  
*Chilomastix* Alexeieff 1911  
*Tetrachilomastix* da Fonseca 1915  
*Copromastix* Aragão 1916  
*Tricercomonas* Wenyon and O Connor 1917  
*Protetramitus* Chalmers and Pekkola 1918

And they may be differentiated as follows —

- A *Without cytostome* —
- 1 With rhizoplast (1) *Protetramitus* Chalmers and Pekkola 1918
  - 2 Without rhizoplast—(2) *Copromastix* Aragão 1916  
(3) *Tricercomonas* Wenyon and O Connor 1917
- B *Cytostome probably present* —  
At all events there is a deep ventral longitudinal furrow—  
(3) *Callodictyon* Carter 1865
- C *Cytostome present* —
- I Trailing flagellum is free —
    - (a) Body dorso ventrally compressed ventral surface with deep depression which serves as a sucker and contains the cytostome and two short free flagella the two thick long trailing flagella issue from this depression—(4) *Costiopsis* Senn 1900
    - (b) Body more or less symmetrical and not compressed or arranged as above with three anterior and one free trailing flagellum—(5) *Tetramitus* Perty 1852
  - II Free trailing flagellum absent —
    - (a) Three anterior flagella—(6) *Chilomastix* Alexeieff 1911
    - (b) Four anterior—(7) *Tetrachilomastix* da Fonseca 1915

Of these genera we are only concerned with *Copromastix* *Tricercomonas* *Chilomastix* and *Tetrachilomastix*

Genus *Copromastix* De Beaurepaire Aragão 1916

Definition.—*Tetramitidinae* without cytostome and rhizoplast

*Copromastix* *prowazeki* Aragão 1916

Found in cultures of human faeces in Brazil

Genus *Tricercomonas* Wenyon and O Connor 1916

Definition.—*Tetramitidinae* without cytostome and with three

B *Habitat intestine of Man in Alexandria* —

Anterior flagellum long and thin cytostomic flagellum shorter and stouter

Size 4-9 microns long but with variable width  $\approx$  4 microns in narrow forms Cysts 4.5-6 microns in length (3)

*Intestinalis*

Only *Embadomonas intestinalis* Wenyon and O Connor 1916 concerns us

*Embadomonas intestinalis* Wenyon and O Connor 1916

Synonym.—*Waskia intestinalis* Wenyon and O Connor 1916

Definition.—*Embadomonas* found in the intestine of man in Alexandria Size 4-9 microns in length but with variable width some 3-4 microns in narrow forms Cysts 4.5-6 microns in length

Remarks.—This flagellate was found by Wenyon and O Connor in two cases in the Orvi el Waskia section of the 19th General

cytostome  
ytostome)  
touter and  
f the cyto



FIG. 75.—*Embadomonas intestinalis* (WENYON AND O CONNOR 1916)  
Showing dividing form flagellate and cyst  
(After Wenyon and O Connor)

The cytoplasm is pale frequently vacuolated with an anteriorly situate nucleus which has a nuclear membrane and a central karyosome. On the surface of the nuclear membrane there are two granules from which the flagella arise

Life-History.—Forms with two cytostomes and four flagella have been seen indicative of division

The cysts are pear shaped bodies of a pearly white appearance and quite structureless unless stained when certain nuclear structures can be made out

Pathogenicity.—There is no evidence that it is pathogenic

## SUBFAMILY TETRAMITIDINÆ Chalmers and Pikkola 1918

Definition.—Tetramitidæ with or without a cytostome with four to six flagella but without an axostyle

Classification.—Eight genera belong to the subfamily—viz —

MONADINA

lows —

length —  
 which resemble *C. mesnili*  
 e—(1) *Caulleryi*  
 length —

about  $7 \times 5.6$  microns in  
 flagella are difficult to see—

ysts large about  $8 \times 6$   
 anterior flagella are very

length —  
 cytosome connected to the  
 plast, size  $9 \times 12$  microns

karyosome and without  
 microns in length—

(5) *Cuniculi*

B Characters unknown to us —

Found in species of *Motella*—(6) *Motella*

*Chilomastix mesnili* Wenyon 1910

Synonyms.—(a) *Cercomonas hominis* Davaine 1869 *pro parte*,  
 (b) *Monocercomonas hominis* Epstein, 1893 *nec* Grassi 1879, (c) *Tri-*  
*chomonas intestinalis* Roos 1893 *pro parte nec* Leuckart 1879  
 (d) *Macrostoma mesnili* Wenyon 1910, (e)  
*Fanapepea intestinalis* Prowazek 1911, (f)



FIG 77—*Chilomastix mesnili*. WENYON 1910

Note the membrane raised by the cytosomic flagellum

**Definition**—*Chilomastix* of medium size with long cytostome and with cysts about  $7 \times 5.6$  microns in which the anterior flagella are difficult to see

**Historical**.—This flagellate appears to have been first noted by Davaine in 1860 being called Form A of *Cercomonas hominis*. After this it was noted by Roos in 1893 and by Epstein in the same year. It was rediscovered and properly described by Wenyon in 1910 and later noted by Prowazek in 1911, Nattan Larrier in 1912, Brumpt in 1912, Gaebel in

**Type Species**—The type and only species is *Tricercomonas hominis* Wenyon and O Connor 1916

*Tricercomonas hominis* Wenyon and O Connor 1916

cytostome could be seen. The cytoplasm contains bacilli and cocci, a nucleus with a central karyosome and a nuclear membrane which is drawn out into a cone like elevation from the summit of which the flagella take their origin.

**Life-History**—As forms with two nuclei have been seen it is presumed that binary division may take place. The cysts are oval (8 microns in length and about half this breadth) containing one to four nuclei of the same type as the flagellate.

**Pathogenicity**—It is believed to be non pathogenic.



FIG. 76.—*Tricercomonas testinasis* WENYON AND O CONNOR 1915

Two flagellate forms (compare side view with Fig. 71) and one cyst (After Wenyon and O Connor from the publications of the Wellcome Bureau of Scientific Research)

#### Genus *Chilomastix* Alexeieff 1911

**Synonyms**—(1) *Cercomonas* DAVIDSON 1884 *pro part* nec Dujardin

**Type Species**—*Chilomastix caulleryi* Alexeieff 1911  
*Macrostoma caulleryi* found in the intestine of tadpoles  
**Other Species**—*C. mesrili* Wenyon 1910, *C. motella* Alexeieff 1912, *C. bitencourti* da Fonseca 1915, *C. capra* da Fonseca 1915, *C. cuniculi* da Fonseca 1915

## II With undulating membrane --

- 1 Three anterior flagella—(8) *Trichomonas* Donne 1837
- 2 Four anterior flagella—(9) *Tetratrichomonas* Paris 1910
- 3 Five anterior flagella—(10) *Pentatrichomonas* Chat terjee 1915

Of these genera *Trichomonas*, *Tetratrichomonas* and *Pentatrichomonas* concern us

Genus *Trichomonas* Donne 1837

FIG. 78 — *Tetratrichomonas galinarum* (MARTIN AND ROBERTSON 1912) ( $\times 2000$  DIAMETERS)

a trace of a parabasal. The nucleus is also represented while the axostyle shows exceedingly clearly. He also saw the cytostome, Bensen (1912) brought into line with the results of Kuczynski and Swezy with the illustration of which a number have been carefully described and drawn by Dobell, Alexeeff, Martin and Robertson, Kuczynski and by Hofoid and Swezy.

elongated posteriorly into a tail. The name was first spelt *Tricomonas* but afterwards altered to *Trichomonas*.

Dujardin (1841) described *T. limacis* from *Limax agrestis* in much the same terms and so did Perty (1852) with regard to *T. batrachorum* though he depicted the axostyle but Stein's figures of Perty's organism show clearly the three anterior flagella, the undulating membrane, the posterior free flagellum, the axostyle, the nucleus and the cytostome and in this way was laid the foundations upon which the main features of the genus were placed.

Returning now to the type *T. vaginalis* this was restudied in 1884 by Blochmann who illustrated the three anterior flagella, the undulating membrane, the axostyle and the nucleus but in the same year Hunstler produced a much better illustration showing four anterior flagella taking their origin from a blepharoplast from which the undulating membrane also arose while this shows

with the illustration of which a number have been carefully described and drawn by Dobell, Alexeeff, Martin and Robertson, Kuczynski and by Hofoid and Swezy.







The only difficulty is with the type *T. vaginalis*. Does it

!

terior granule arises a posterior flagellum which passing backwards forms the undulating membrane and finally terminates in a free posterior flagellum like striated undulating

to this end springing from the blepharoplast and directed posteriorly over the nucleus is a peculiar body clear with bounding lines which projects from the posterior end in a spine and often contains chromatinic granules. This organella is the axostyle called also the *baguette interne*. It may be a supporting structure but it may also be concerned in movement. It does not stain with nuclear stains and therefore appears clear while in the living

axostyle leaves the cytoplasm

**Life-History**—Binary division with its nuclear changes have been carefully studied by Kofoid and Sweeney. Multiple fission has also been described. Transference from host to host is by the typical cysts.

**Classification**—A very large and increasing number of species of doubtful value are in existence—e.g. *T. baettrachorum* Perty 1852 in frogs, *T. ...*

have also described forms in fowls and Kuczyński has contributed an elaborate paper on the morphology of the genus.

Four species are said to occur in man—*T. vaginalis*, *T. hominis*, *T. dysenteriae* and *T. pulmonalis*.

While some differences do exist in the animal forms still there is nothing of a specific nature to be found in the human and it is quite possible that they are all one species and that they are the same as that found in

flagella unless but on this point wrong with it

long loose folds as well as a thinish axostyle and a nucleus rich in chromatin and bounded by a very definite membrane. He named this organism *Trichomonas prowazeki* but as it possesses four and not three anterior unequal flagella Parisi created a new genus with it as the type.

**Tetratrichomonas vaginalis** Castellani and Chalmers 1919

Kunstler described *Trichomonas vaginalis* with four flagella and we have seen forms in the human vagina with four flagella.

**Tetratrichomonas intestinalis** Chalmers and Pekkola 1919

Found in human faeces in Egypt by Wenyon and O'Conno and later by Chalmers and Pekkola in the Sudan.

**Genus Pentatrichomonas** Chatterjee 1915

with a cytostome and undulating  
*ardindeltshi* Derrieu and Ray  
 of man in Africa and India  
 this genus for a flagellate which  
 in Bengal and which at the time  
 he found in man  
 be called *P. bengalensis*. This organism agrees in most particulars  
 except measurement with that mentioned above under the heading  
*Hexamastix* Derrieu and Raynaud and therefore Chatterjee's  
 generic name takes the place of this *Hexamastix* but Derrieu and  
 his name has priority and the parasite becomes

to be present in thirty two cases.

*ardindeltshi* (Derrieu and Raynaud 1914)

## REFERENCES

- and regard to the Tetramitidae with a view to  
 For references see Feld H H (1912) *Bibliographia Protozoologica Archiv f. Protistenkunde* xxvi p 444 Jena  
 DOPLEIN AND KORHLER (1912) Ueberblick über Stamm der Protozoen  
 Kolle and Wassermann's Handbuch der Pathogenen Mikroorganismen  
 Jena  
 MINCHIN (1912) *An Introduction to the Study of the Protozoa* London  
 PROWAZEK (1911) *Handbuch der Pathogenen Protozoen* Leipzig

*Trichomonas dysenteriae* Billet 1907

The T. L.

It lives on bacteria, (3) stage of free existence when it is a flagellate. He considers that *Loeschia undulans* Castellani 1904 is allied to this species although Castellani is inclined to believe that it is a stage of an *Oicomonas*.

*Trichomonas pulmonalis* Schmidt 1895

This form has been found by Schmidt and St. Arvult Leyden and Jaffe in the sputum and lungs of persons suffering from phthisis gangrene and putrid bronchitis.

Other Species.—*T. batrachorum* Parry 1852 in the cloaca of *Rana* *tempora*.

W. D. C. in the cloaca of *Lacerta agilis* *T. canis* Davaine in the large bowel of guinea pigs, but this last may perhaps be separated off into a separate genus *Trichomastix* with one long flagellum directed across the body. *T. perronciti* Castellani 1907, in monkeys suffering from diarrhoea is very similar morphologically to *T. hominis*. *T. columbarum* Pro wazek and Aragio 1909 is found in the buccal mucosa of pigeons. Plimmer has shown that flagellates of the type of *Trichomonas* can be found in the blood of snakes.



FIG. 79. FLAGELLATE OF TRICHOMONAS TYPE FOUND IN THE BLOOD OF A LEOPARDINE SNAKE.

*Tetratrichomonas* Parry 1910

Definition.—T. L.

with  
membr.

Tyl.

and t.

(mer.)

salamanders

Remarks.—Alexieff (1909) found a trichomonas-like parasite in the terminal intestine of *Salamandra maculosa* *Triton cristatus* and *Alytes obstetricans* and subsequently in *Hamopsis sarguisuga*. The parasite, measuring 10 to 14 by 4 to 7 microns possessed four free unequal anterior flagella and an undulating membrane thrown into



## Classification Tetramitida and Chilomastix

CHALMERS AND PEKKOLA (1918) *Annals of Tropical Medicine and Parasitology* (reference) xi 3 213 262 Liverpool

## Oicomonas

SAVILLE KENT (1880-81) *Manual of the Infusoria*

SENN (1900) *Engler and Prantl Pflanzenfamilien I* 14 141 147 Leipzig

## Prowazekia

CATELLANI AND CHALMERS (1910) *Annual Meeting of the Far Eastern Association*

WHITMORE (1911) *Archiv für Protistenkunde* xvii 170 Jena

## Euteromonas

CHALMERS AND PEKKOLA (1918) *Journal of Tropical Medicine and Hygiene* July 15 (reference)

## Embademonas

MACKINNON (1910) *Parasitology* vol 1 pp 245 253 (1911) *ibid* vol 10 pp 28 38 (1912) *ibid* vol 5 pp 175 189 (1913) *Quarterly Journal of Microscopical Science* vol lxx pp 227 308 (1914) *ibid* vol lx pp 459 470 (1915) *ibid* vol lxi pp 105 118 London

## Cercomonadida

D JARDIN 1913 1 17 + " "

FOR

" 1

Arch

WE  
WE



- 5 *Toxoplasma* Nicolle and Mancaux 1908
- 6 *Piroplasma* Patton 1893
- 7 *Achromaticus* Dionisi 1898
- 8 *Histoplasma* Dublin, 1906

Unfortunately there has been much confusion with regard to these genera due to lack of certain knowledge with regard to the morphology and life histories of the type species. The controversy was keenest with regard to the points as to whether *Herpetomonas* and *Leptomonas* were or were not the same genus, whether *Herpetomonas* and *Crithidia* are good genera or simply stages in the life cycle of a trypanosome. Briefly the position is this. In 1891

and the flexibility of the body in *Herpetomonas* but modern research fails to confirm the presence of this contractile vacuole in *Herpetomonas* and flexibility *per se* is insufficient to separate the two genera. Prowzek, however, in 1904, described *Herpetomonas* describing *H. muscæ domesticae* as possessing two flagella united by a membrane and arising from a flagellar situated diplosome, but Patton in 1909 and Mickinnon in 1910 have demonstrated

an axostyle. Therefore it would appear that the two genera are indistinguishable and might therefore be united and if so the older name *Leptomonas* would by the law of priority come into use to the exclusion of the name *Herpetomonas* and this may happen but it cannot be adopted at present because the type species of the genus *Leptomonas*—namely *L. butschlii* Kent 1881 is yet been

another

With regard to the controversy as to whether there is a genus *Crithidia* or not the answer is much simpler. There can be no doubt that crithidia like forms exist in the life cycle of many trypanosomes but the work of Patton, Peter and Swingle has clearly shown that there is a separate genus *Crithidia*. Later 1912 named Patton. Further we believe that Miss Peter is correct when she states that *Crithidia* should be placed in the family Trypanosomidae on account of the presence of an annular membrane.

With regard to *Leishmaniasis* and other kinetoplast life-histories



The Herpetomoninæ are distinct forms and are not stages in  
 paper O Farrell in 1913  
 tick *Hyalomma aegyptium*  
 by *Crithidia hyalomma* Usually the flagellate stage is passed in



FIG 81.—THE LIFE CYCLE OF *Crithidia hyalomma* O FARRELL 1913  
 (After O Farrell)

It demonstrates hereditary infection. The arrows in the infection of the ovum except the two on the right hand side are wrong. The left upper arrow should run from the adult to the ovum the other two arrows should be omitted.

haemocœlic fluid of the tick but about the time of and during

These five genera may be differentiated as follows —

(a) Undulating membrane absent —

Genera *Herpetomonas* *Leptomonas* *Leishmania* *Hemphysalis* (Probably all belong to one and the same genus *Herpetomonas*)

(b) Rudimentary undulating membrane present — Genus *Critidia*

**Morphology**—The flagellate stage of the Herpetomoninae is usually an elongated spindle-shaped mass of cytoplasm composed of an inner granular endoplasm surrounded by a periplast (ectoplasm). In the cytoplasm lies a chromatinic mass the tropho-

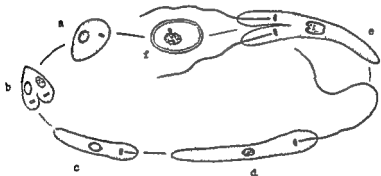


FIG. 90.—THE LIFE HISTORY OF A HERPETOMONAS (After Gantham)

of the intestine of the arthropoda but some live in plants

the  
in  
clea  
a new host

In the new host it appears as a non-flagellate binucleate rounded form often called a Leishmania like body or the Leishmaniform stage which develops in due course into the flagellate stage again

Thus in the life-cycle there are the following stages —

- 1 The flagellate stage
- 2 The post flagellate or encysted stage
- 3 The Leishmaniform stage
- 4 The flagellate stage again

of sexual reproduction

Other Observations

given for *Critidia gerridis* (vide p. 367). He further supports this life cycle by his previous description of a *Herpetomonas* in *Culex pipiens*.

*Herpetomonas jaculum* Léger 1902

fection, and now the ingested cysts which are small oval bodies, with tropho and kineto nuclei form the preflagellate stage of the life cycle, which passes

late forms penetrate

*H. böttschli* Saville Kent 1881

In *Trilobius gracilis*, but it has not been perfectly studied

*H. pycnosoma* Roubaud 1904

In *Pycnosoma putorum* Wiedmann in Africa

*H. davidi* Lafont 1909

Described in *Euphorbia pulchifera* by Lafont, in Mauritius, Ceylon and Réunion, East and West Africa, West Indies, Portugal and India. We have

resembles that described by Porter for *Crithidia melophagia* in the fly *Melophagus ovinus*

**Pathogenicity**—In an able paper published in 1916 Tenthum and Porter have shown that by feeding and by inoculation of various forms of herpetomonads and crithidias acute and chronic attacks of herpetomoniasis can be induced in vertebrates. Thus *Herpetomonas jaculum* from *Nepa cinerea* by feeding produced the disease in mice birds a snake frogs newts and fish *H. stratiomyia* from *Stratiomyia chameleon* in a mouse *H. pediculi* from *Pelliculus corporis* in mice *H. culicis* from *Culex pipiens* in birds

in man is from aquatic insects and per os. Experimental herpeto-

day and that as the result of change of habitat (brought about by invasion or insect fecal contamination of a bite) a herpetomonas may find itself in a vertebrate host and there taking on its fish-maniform stage becomes pathogenic producing the disease herpetomoniasis which may be acute or chronic. In the acute disease the flagellata form

infection may

herpetomonad  
in Algeria in

with Oriental sore was endemic and suggested that the possible carrier was a phlebotomus. In the same year Chatton and Le Blanc found fishmanina like forms in the red blood cells of geolops in Tunis.

In 1915 Bayou found herpetomonas in the alimentary canal of *Chameleon pumilus* at Robbin Island and also in a fly *Scatophaga tentata* and suggested the possibility of infection of the vertebrate by swallowing a fly.

In 1914 Linney suggested that the oral fishmaniasis of Paraguay might have its reservoir in rattlesnakes and its carrier in ticks or Simulium flies.

On the other hand Archibald as possible not readily differentiate

the parasites of Sudan from those of the Mediterranean



often found this species in Ceylon. The parts of the plants affected are not healthy and the diseases called 'flagellosis'. Miss Robertson has found a similar parasite in cotton plants in Uganda. It measures 10.5 to 15 microns.

Other Species.—*H. gracilis* Léger, 1902, in the Malpighian tubules of the

### Genus *Crithidia* Léger 1902, emendated Patton 1907

**Definition.**—Herpetomonadæ, in which the schizont is characterized by an attenuated posterior end, to which the flagellum is attached by a rudimentary enveloping membrane. The kinetonucleus is situated close to the trophonucleus either on the flagellar sides or slightly and rarely a little on the flagellar aspect of this structure.



FIG. 84.—DIAGRAM OF A CRITHIDIA

**Remarks.**—In 1902 Léger created this genus for a flagellate organism which he found in the alimentary canal of *Anopheles maculipennis* and in 1907 Patton worked out the life history of *Crithidia gerridis*.

### *Crithidia gerridis* Patton 1907

This flagellate is a parasite in *Gerris fossarium labriculus* and in a species of *Microvelia* and in a water bug allied to *Perillopus* found in Madrid. The

The kinetonucleus increases in size and the trophonucleus shows its chromo-

on  
are

cc  
mu

nu  
lu\*

- I It is readily infecting dogs
- II It causes a local cutaneous lesion when inoculated into the skin of a monkey

He further believes that it is a special variety of *L. donovani* because —

- I It has signs of a coracal stage in its life-history. This has been objected to by Wenyon and Laveran but has been confirmed by Smallman in two cases from Malta and by Stathan and Butler in Serra Leone
- II Experimental evidence is not in favour of its being insect borne. On the contrary, it is found in the blood of man and monkey.
- III The local lesion produced by intracutaneous inoculations into monkeys does not exhibit any eosinophile leucocytes which is different from the lesions produced by *L. tropica* but it is not known whether this occurs or not in those due to *L. donovani* and *L. infantum*.

We therefore recognize it as *L. donovani varietas archibaldi*

### *Leishmania donovani* Laveran and Mesnil 1903

**Definition** — *Leishmania* producing in man the signs and symptoms of tropical kala azar in experimental monkeys general and local infections but not readily infecting dogs

**History** — The history is fully given in Chapter XLVII p 1289 and it need only be remarked that the parasite discovered by Leishman in 1900 was described by himself and by Donovan in 1903 while Rogers in 1904 cultivated the parasite at 22° C and discovered the flagellate stage

Christophers in 1904 considerably added to our knowledge of these parasites and Patton in 1906 and 1907 showed that they were

apparently more in the form of a natural culture than of a cyclic development

**Development in the Bug** — According to Patton the parasites are ingested by the bug enclosed in the large cells or leucocytes as just mentioned and develop into fully flagellated forms without reference to the temperature of the external air

The first change begins usually by an increase in size up to 4 to 7  $\mu$  and a vacuolation of the cytoplasm on the second day but may be deferred for several days

The single parasite may proceed directly to flagellation by the appearance of an area stained bright pink by Giemsa and called the flagellar vacuole. This vacuole which has a dark centre rapidly increases in size up to 1 to 3  $\mu$  and passing to the surface sends out a small pink brush which forms the flagellum by merely growing longer. There appears to be no doubt that the

*Crithidia melophagia* Flu 1908.

Flu has described *Crithidia melophagia* in *Melophagus ovinus*, a parasite of

Genus *Leishmania* III Ross 1903

Synonyms—*Piroplasma* Laveran and Mesnil, 1903 *Helcosoma* Wright 1903 *Herpetomonas* Rogers 1904

Definition—Herpetomonadæ living principally in endothelial cells, but also found in leucocytes and in the peripheral blood of mammals as small oval cytoplasmic masses with tropho and kineto-nuclei and developing into flagellate bodies in cultures

Remarks.—Three species are known in man morphologically similar but pathogenetically different *Leishmania donovani* Laveran and Mesnil 1903 *L. infantum* Nicolle 1908 and *L. tropica* Wright, 1903 and probably there are other varieties

With regard to these species there is a general consensus of opinion that *L. tropica* with its variety *americana* is distinct from *L. donovani* and *L. infantum*. In respect to the two last named forms it has been argued that they are identical because—

- I Both attack adults and children
- II A monkey immunized against *L. infantum* is refractory to *L. donovani*

On the other hand there are some differences viz—

- I It is true that both attack adults and children but the latter are much more easily infected by *L. infantum*
- II *L. infantum* infects dogs readily while these are more refractory to *L. donovani*
- III *L. infantum* produces a local cutaneous lesion with or without a general infection when inoculated into or under the skin

For the present we shall treat *L. donovani* and *L. infantum* as separate parasites

The Sudan parasite is considered by Archibald to be distinct from *L. infantum* because—



distinguish it from other forms of infantile splenic anemia. In 1907 Nicolle and Cassuto observed the parasites in the spleen of a child in Tunisia suffering from irregular fever splenomegaly etc and Nicolle named this disease infantile kala azar which is a most suitable name. After this it was described in Crete in 1907 by Archer in Sicily Stromboli and Calabria by Gabbi and Teletti in 1910 it was found in Malta by Critien in Lisbon by Alvares while Gabbi proved that the disease ponos as seen in Spezzia was the same disease and Christomanos Aravandinos and Michaelides found it in the Grecian islands and Greece itself. In 1911 Christomanos found it in several places in Greece and Asia Minor while Batinos found it in Corfu Kefalinos in Paris and it was found to be widely distributed in Southern Italy and Sicily. Martzinowsky has observed cases in Moscow Tashum Ibrahim in Tripoli Lemaire in Algiers and Sluka and Zarfi in Tashkent in Turkestan Marshall reports the disease among children of about twelve years of age in the Sennar province of the Sudan.



FIG. 87.—*Leishmania infantum* NICOLLE FROM THE LIVER OF A DOG EXPERIMENTALLY INFECTED (From a microphotograph by Basile)

It is thus seen that *L. infantum* is mainly found around the Mediterranean basin but may extend to Moscow and to Turkestan probably its geographical distribution is but little known at present.

The study of the life history began by the experiments of Nicolle who in 1908 successfully inoculated a dog in Tunis intrahepatically and intraperitoneally with splenic blood from a case of infantile kala azar monkeys were also inoculated successfully.

Later Manceaux Comte Laveran Pettit Jemna Di Cristina Cannata Alvares da Silva Pulvirenti and Tomacelli successfully inoculated dogs monkeys and guinea pigs and Volpino

produced a purely local lesion somewhat analogous to Oriental sore by inoculating the corner of a rabbit from an infected dog.

In 1908 Nicolle and Comte recorded the discovery of spontaneous kala azar in dogs in Tunis and eventually found 18 per cent to be infected in the spring but this percentage was raised by the Sergeants to 72 per cent in the summer in Algiers and Sevenet has found that in Algiers 16 per cent show infection in the spring and

fact when he found infected dogs in every house where kala azar had been found by Gabbi and still more interesting is the observation that infected dogs were found in houses without cases of kala

flagellum forms in this vacuole, and is not directly connected with the kinetocytocyst.

The flagellate form has a dark blue, granular cytoplasm with a circular kinetocyst which stains deeply in the centre, and a kinetocyst nucleus lying



FIG. 86.—*Leishmania donovani* LAVERAN AND MESNIL.

Free ...  
 cytes ...  
 show ...  
 para ...  
 Patten ...  
 in the ...  
 the de ...

acros ...  
 a ...  
 int ...  
 dis ...

liver and spleen. Control dogs were then killed and found healthy. Basile concludes that *P. serraticeps* is the carrier of the disease. In Bordonaro he examined 1000 fleas from dogs and the beds of families but found only four infected with *Leishmania*. With regard to *P. irritans* Basile finds that it is frequently a parasite of the dog and that among specimens caught in the bed of a child suffering from kala azar one was found to contain *Leishmania*. From experiments he believes that fleas are infective from December to March. There is one curious point noted that *Leishmania* was found in spleen, liver and bone marrow only a few days before death though the fleas had bitten the dogs three months earlier.

With regard to the objections to this work Gabbi has pointed out—

1. Canine and human fleas placed in contact with pure cultures of *Leishmania* on Nicolle's blood agar show blood in the gut but no *Leishmania*.
2. *Leishmania* in culture with intestinal bacteria from the flea or in culture with the juice from the same do not develop.
3. Starving fleas placed in contact with spleen juice obtained by puncture from a child with kala azar do not become infected with *Leishmania*.

in the flea

- F
- Crithidia pulicis* Wenyon 1908 in *Xenopsylla cleopatrae*  
*Crithidia stenophthalmi* Patton and Strickland 1908 in *Ptenophthalmus aegyptus*  
*Crithidia hystrichopsyllæ* Mackinnon 1909 in *Hystrichopsylla talpæ*  
*Crithidia pulicis* Porter 1911 in *Pulex irritans*  
*Herpetomonas stenophthalmi* Mackinnon 1909 in *Ptenophthalmus aegyptus*
- There are also a number of unnamed flagellates recorded—e.g. a species of

azar but subsequently a case has already occurred in one of these houses. Canine kala azar has also been found in Catania slightly in Rome in 6.69 per cent of dogs in Greece by Cardamatis in 2.66 per cent in Lisbon also in Malta by Critien and Babington and in a few dogs in Colombo by Castellani in 1911. It must be noted

we have found a four month old kitten to be infected in Algeria. It will thus be seen that of all the endemic centres of infantile kala azar Palermo alone affords no evidence of natural canine kala azar.

As the result of his work Basile supporting Nicolle has come to the conclusion that infantile and canine kala azar are one and the same disease. He reared a number of dogs in the laboratory in Rome (where canine kala azar is rare) and some of these he took to Bordonaro where they contracted canine kala azar and died. The parasites were found in the bone marrow, spleen and liver (Fig 87) and also in *Pulex serraticeps* taken from them during the last stages of the disease. The dogs in Rome were subsequently killed and found to be free from *L. infantum*. *P. serraticeps* from a laboratory dog whose bone marrow contained no

portions—one was used for smears and the other was made into an emulsion and injected subcutaneously into a young dog one month old the bone marrow of which had been shown to be free from *Leishmania* while another dog was used as a control.

The smears from the infected fleas showed numerous specimens of *Leishmania* in a state of multiplication while the control fleas were free. After fifteen days the dog became ill with fever and loss of appetite and dejection and showed the parasites in the peripheral blood. In twenty nine days the dog died probably as a result of an operation to obtain bone marrow from the tibia. Natural infection by flea bites was effected by introducing a sick dog covered with fleas into a cage containing a bitch and two thirty day old puppies whose bone marrow had been found free from infection. In thirty days these dogs were found to be infected

of people living in Bordonaro and were fed upon laboratory reared

marrow showed large numbers of mononuclear cells with parasites. The heart muscle showed cloudy swelling. No parasites are found in the lungs.

There is a growing suspicion that this is quite distinct from any human disease because—

- 1 . . . . .
- 2 . . . . .

It is known to occur in Africa, Europe, and Asia, but not in America or Oceania.

The formation of a local sore on the cornea of a rabbit must be remembered as indicating the possibility of a local disease like that caused by *L. tropica*.

#### *Leishmania tropica* Wright, 1903.

if this is so, the correct name of the parasite should be *L. julliaculosa* Firth 1891.

It was specially investigated in 1905 by James in Delhi sore, Lahore sore, and Frontier sore. Nicolle has obtained cultures of the parasite on the McNeal-Novy medium and Carter describes sexual forms. Carter, Balfour, and Nattan-Larrier believe, in our opinion correctly, that there are several varieties or species included under the term *L. tropica*, thus Carter maintains that the Cambay sore is different from the 'clou de Gafsa' of Africa. Werner, Carini, and Splendore have shown that the sore may spread to or begin on mucous membranes.

**Distribution.**—It is found principally in Asia in India, China, Asia Minor, Persia etc., but also occurs in Africa, Algeria, Tunisia, on the Niger and in the Egyptian Sudan, in Mexico, Panama and South America.

carrier

**Morphology**—*L. infantum* so closely resembles *L. donovani* a

Neal  
1 m  
the

agar. It can be subcultured indefinitely. No distinct differences can be discerned between the three species of *Leishmania* in culture. *L. infantum* can to a certain extent be distinguished from *L. donovani* by the fact that the latter is less easily inoculable with success into dogs.

**Pathogenicity**—It is the cause of infantile kala azar and at one time was considered to be the cause of canine kala azar which we will now describe.

**Canine Kala Azar**—There are two types of canine kala azar in

young dogs when it is  
attent type followed by

appetite wasting tremors motor disturbance in the hind limbs and rarely diarrhoea. The animal finally dies in a comatose condition at the end of three to five months.

Chronic canine kala azar begins without any apparent symptoms except perhaps loss in weight but as it progresses anaemia sets in and tremors together with motor disturbances of the hind limbs

increased and parasites occur in the endothelial cells. Parasites may be found in the round celled infiltration under the capsule of the kidney. The suprarenal bodies show cloudy swelling of the cortical cells infarcts in the medulla and vacuolation of the cells which may be invaded in patches by the parasites. The pancreas showed a hypertrophy of the connective tissue with the presence of the parasites in the endothelium of the lymphatic. The bone-

good for *Stegomyia fasciata*. Phlebotomus is suspected but there is no experimental evidence. Flu suspects ticks as being the possible carriers in Dutch Guiana. Pediculi have no supporters and *Stomoxys* is not regarded as a likely carrier. As Patton has

and on mucous membranes in Asia, Africa and America and which are commonly known as Oriental sore but which also have a large number of local names such as pian bois, espundia, Delhi sore, Bagdad button, clou de Gafsa, ulcer of Bauru etc.

typical characters

#### SUBFAMILY TRYPANOSOMINÆ CASTELLANI AND CHALMERS 1919

**Definition**—Trypanosomidæ in which the kinetonucleus is situated in certain stages of the life cycle between the trophonucleus and the flagellar extremity of the body. A well developed undulating membrane is present.

**Type Genus**—*Trypanosoma* Gruby 1843 *emeri* Davit Laveran and Mesnil 1901

**Classification**—A brief history of the discovery of the more important species of this family has been given in Chapter I. The genera which have been described are—

1901  
4

But *Trypanoplasma* and *Trypanophis* belong to the Bodonidæ and not to the Trypanosomidæ (*vide p. 337*)

*Endotrypanum* would appear to be an immature trypanosome without an undulating membrane and parasitic in red corpuscles. In 1905 Nissle drew attention to the occasional invasion of red cells

first classified as a  
became known it  
under a separate

*Trypanosoma* of

fifteenth day and these can be repeated apparently indefinitely. Forty five generations have been recorded during a period of eighteen months.

It is usual to state that there are no differences between the cultural forms of *L. tropica* and *L. donovani* but Row considers that there are several points of difference—viz that the flagellate forms of *L. tropica* are longer and larger than the flagellum is

successfully inoculated from cases of Oriental sore. The incubation period varies from sixteen days to six and a half months and the papule is generally ushered in with febrile symptoms lasting several days. The inoculated sore begins as a papule and becomes a nodule when excised and examined it presents the typical appearances of Oriental sore and contains *L. tropica*.

Successful inoculations are recorded from man to monkeys and from dogs

is complete cure and the produced is a leishmaniasis.

It is dogs affords immunity against *L. tropica* during and after the attack. Oriental sore protects monkeys partially or completely against *L. donovani*.

**Insect Carriers**—No insect at present has been demonstrated to be the true host of *L. tropica*. *Musca domestica* may possibly be a carrier because the parasites can retain their vitality therein and may be transferred to any raw surface and thus induce infection. But it is not a true host though Row has found the contents of the gut to be infective and believes that infection can be spread by its feces. There can be no doubt that a natural culture produces flagellates and, according to Patton even post flagellate forms can take place in the bed bug *Cimex retundatus* but all attempts at transmission have failed and the same condition of affairs holds



which ends in a little bead—the blepharoplast—from which the flagellum which is also composed of chromatium arises. A more primitive arrangement is for the kintonucleus to contain the blepharoplast, which is really only a centrosome. Under these circumstances the flagellum will arise from the kintonucleus. When the blepharoplast is separate from the kintonucleus it is a moot point as to whether there is or there is not another centrosome in that nucleus.

The flagellum runs outwards through the endoplasm to the ectoplasm. In this course it presents three portions (1) The root in the endoplasm (2) the undulating portion in the ectoplasm and (3) the free portion. In some stages of the life history the flagellum instead of turning along the undulating membrane projects from its blepharoplast through the endo and ectoplasm to the outside of the parasite.

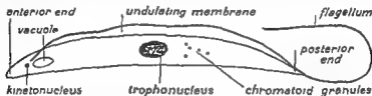


FIG. 88.—DIAGRAM SHOWING THE STRUCTURE AND POLARITY OF A TRYPANOSOME.

The undulating membrane therefore is the layer of ectoplasm (periplast) raised from the surface of the parasite by the second portion of the flagellum along a line sometimes called dorsal and may be prolonged a certain distance along what is generally considered to be its free portion. The membrane is variously described as being homogeneous or strengthened by myonemes as in *Hæmophilus proteus noctua*.

Besides these structures the endoplasm often contains a vacuole looked upon by some observers as a contractile vacuole while others deny its existence. There is no doubt about its existence in *Trypanosoma castellani*. Chromatoid granules can also be seen in the cytoplasm.

Before leaving this part of the subject it must be noted that many authors have given various names to the trophonucleus, kintonucleus, centrosome and blepharoplast. We use the terms in the same sense in which they have been used by Minchin and Woodcock—viz: the principal nucleus is a trophonucleus because it is believed to be largely concerned in nutrition; it contains an achromatic body which is the centrosome. The smaller nucleus is a kintonucleus because it is mainly concerned in motion, while the little bead connected with the flagellum is looked upon as a

---

**SUBFAMILY TRYPANOSOMINÆ**

which we shall presently suggest a classification (*vide*  
and the genera *Endotrypanum* *Schizotrypanum* and *Rhimonas*

***Trypanosoma* Gruby 1843**

**Synonyms** — *Imarba* Myer 1843 *Paramacium* May  
*Globularia* Wedl 1850 *Undulina* Lankester 1871 *Herp*  
Kent 1878 *Hamatomonas* Mitrochin 1883 *Trypan*  
Danilewsky 1885 *Trypanosom* Luchic 1906

**Definition.**—Trypanosominae with the periplast raised  
longitudinal undulating membrane along which the single  
runs

**Historical**—The history of the genus has been largely  
in Chapter I and we need only remind the reader that it  
by Valentin in 1841 reporting minute bodies in the  
*Salmosia* Linnæus the brown trout which induced Glug  
forward his discovery of *Trypanosoma sanguinis* in 1843

them  
" " " "

considerably in different species and may even be rounded

*In the Vertebrate*—If an uninfected or clean non immune is infected by the bites of infective invertebrates at first no parasites are to be found in the peripheral blood and some days must elapse before they appear. This interval is the incubation period. What takes place during this period is but little known the only observations being those by Fantham who saw a few rounded forms fifteen



FIG 89.—DIAGRAM OF THE LIFE CYCLE OF *Trypanosoma lewisi* SAVILLE & HENT 1880 IN THE BODY OF THE RAT

—'op

of

blepharoplast because it is an achromatic body connected with a cilium or a flagellum

We now come to a point on which there is a great difference of opinion—viz the polarity of the parasite. There can be no doubt

now and blood being the method of propulsion must be morphologists (Woodcock) no doubt that at times the

membrane while movements have been described by Tullach and Tullach. In this work

the word, therefore, means the non flagellated end of the trypanosome

in various some way along the flagellum

It has already been noted that the body of the parasite is slightly compressed laterally and the edge with the undulating membrane is considered to be dorsal. In some species a supporting structure somewhat of the nature of an axostyle has been described.

The measurements of the parasite are from the non flagellate extremity to the kinetonucleus from that to the interior end of the trophonucleus from that to the posterior end of the trophonucleus from that to the tip of the flagellum the sum of these giving the length, while the width is taken in the region of the trophonucleus.

Strother and others

inserted and the first removed. The transparent paper is again rotated to take in another portion of the axis of the parasite and this is repeated until all the deviations of the parasite have been followed and the distal extremity reached.

The results of careful measurements have been to show that some trypanosomes are polymorphic and others are not.

Food is absorbed by osmosis from the liquid in which the parasite is living.

**Life-History.**—The life history of a trypanosome is not as yet fully known but it is recognized that it has an alternation of generations associated with an alteration of hosts, one generation being usually completed in the blood of a vertebrate and the other in the alimentary canal and its appendages of some blood-sucking invertebrate.

■ an opposite pole

w trophonuclei are

formed

*Rosette Formation*—The medium sized parasite according to Moore Breinl and Hindle grows into large forms which pass through the following development —

x The trophonucleus undergoes reduction by amitosis the re

ich travels—increasing with which it perhaps

fuses but this is not definitely known

In *T castellani* a strand forms between the kineto and tropho nuclei instead of this travelling body which ■ seen in *T lewisi* and *T equiperdus*



FIG 90—*Crithidia melopha* a FLU  
(After Flu)

1 Microgamete 2 macrogamete 3 zygote 4 ookinete and degenerating microgamete 5 ■ ookinete in the alimentary canal and ovary

3 The tropho and kineto nuclei divide to form fusion masses consisting of two four seven or more small parasites which at first possess only the old flagellum but in which later new flagella form (the rosette formation) This stage is not definitely known in *T castellani*

When the parasites are present in the peripheral blood they usually show a marked pleomorphism. Thus in *T. castellani* Miss Robertson finds short forms 14 to 20  $\mu$  in length and long forms 20 to 24  $\mu$  in length and long forms

place by schizogony, with or without entering an endothelial or other cell. The number of trypanosomes in the blood varies considerably from time to time, apparently in more or less regular cycles, and their disappearance appears to be associated with the encystment in the lungs, spleen, bone marrow, etc. in the form of the latent bodies described by Breinl, Fantham, and others. During their life in the vertebrate it was, at one time, thought that they could propagate their species—in part at all events—by granules which are comparable to the infective granule described by

ever untraced in trypanosomes.

There can be no doubt that there is a reaction on the part of the cells of the vertebrate against the trypanosomes with the formation of antibodies in the form of trypanolysins, etc., but more will be said on this subject later.

**Binary Fission.**—A binary fission takes place with or without growth. This is brought about by amitotic division of the kinetoplast and trypomastix nucleus followed by the formation of a new flagellum in the daughter parasite and the division of the cytoplasm.

The following figures

- 1
- 2
- 3 The band elongates and divides into two portions.
- 4 The two portions move apart, all trace of the vesicle disappears and the two new kinetoplasts are formed.
- 5 The blepharoplast divides at the same time as the kinetoplast.
- 6 Either the old flagellum divides or a new flagellum develops from one of the new blepharoplasts. The process varies in different species.
- 7 The central karyosome of the trypomastix nucleus either divides and the two portions move to opposite poles of the nucleus, but are connected by a fine line, or the chromatin forms an equatorial plate.

the cyst which is formed by the periplast of the original trypanosome, and enter red blood cells in which they develop into sexually differentiated trypanosomes—*i.e.* females with one and males with two nuclei. These are the forms which infect the *Lanus* or invertebrate host and do not multiply in the vertebrate host.

**Method of Transmission**—The blood of the infected vertebrate is not always infective for the invertebrate host. Thus Miss Robertson has shown with regard to *T. castellani* that the tsetse fly cannot be infected by feeding just before an outburst of multiplication in the vertebrate host or during the period of destruction which precedes a paucity period or at the summit of an exalted period or during certain periods of rapid multiplication when the absolute and relative numbers of the short forms mentioned above are diminished.

Immediately after infection the invertebrate host can *mechanically* convey the infection to a *clean* host and this power persists for about twenty four hours after which the invertebrate host becomes non infective and remains so for a varying period which was found by Kleine not to be less than eighteen days as regards *Glossina palpalis* and *T. castellani* and by Kinghorn and Yorke to be about fourteen days in *G. morsitans* infected with *T. rhodesense*.

fective period cannot possibly be classed as mechanical. The fact

trypanosome undergoes part of its life cycle in the invertebrate host and the first question which naturally arises is the fate of the so called male and female forms found in the blood of the vertebrate. Are they true male and female forms and do they conjugate and form an ookinete or zygote or do they not?

**Conjugation**—It is difficult to be certain that conjugation has been seen and not division. It has been described by Keysselitz in *Trypanoplasma* infected with *Trypanoplasma* rats infected with *T. lewisi*, which reference will be made

On the other hand careful observers like Miss Robertson and Captain Patton have quite failed to see this process in their studies of trypanosomes and herpetomonads. The conclusion is that conjugation has not been proved to be present in trypanosomes so far.

**Development without conjugation**  
there is no conjugation  
the invertebrate host  
and may be classed as follows ---

blood sucker would conjugate and produce ookinetes and perhaps oocysts from which forms would be produced which might infect the proboscis of the same individual or by entering into the eggs infect a new generation which alone might be the means of dissemination of the parasite. But these theoretical views have so far not been confirmed by actual observations which must now be discussed seriatim.

**So-called Sexual Forms**—According to Prowazek *T. lewisi* can be differentiated into three forms—(1) male (2) female (3) in different and according to Prowazek Luke Necht and Mayer the same can be seen in *T. castellanii* but according to Holmes only male & females and young females can be seen in *T. castellanii*.

**Male Forms**—These are defined to be very slender trypanosomes actively motile with an elongated nucleus which stains well.

**Female Forms**—Broad sluggish trypanosomes with reticulated protoplasm and a round nucleus both of which stain poorly. They possess a slender undulating membrane and a short flagellum.

**Indifferent Forms**—These are the forms most commonly met with

of these may be simply the ordinary trypanosome in various stages of growth and division as described above.

Miss Robertson has probably arrived at the truth when she says that the short forms (13-20 microns) of *T. castellanii* the so-called female forms are really the adults which by growth become the indifferent forms which are merely steps in the formation of the slender forms so-called male forms which are the dividing stage of this trypanosome.

Chagas has shown that *Schizotrypanum cruzi* in the lungs may lose its flagellum and become curved into an arc the extremities of which fuse forming at first a ring which subsequently becomes a sphere with a trophic nucleus and a kinetocore nucleus the latter of which is expelled in female forms while it is retained in male forms. In this manner the microgametocytes and the megagametocytes arise. Each of these divides into eight microgametes which are unipolar and eight megagametes which have a trophic nucleus in kinetocore nucleus united by a filament. These gametes escape from



*D Salivary Gland Infection*—According to Bruce and his collaborators if *G palpalis* is fed with *T castellani* the proboscis is not involved in the further development. The fly now becomes non-infective for some twenty-eight days on an average. The

— 26 to 28 days disappear (possibly become intracellular forms already mentioned above) and now the fly is found to be infective and to remain so for long periods. These short stumpy forms have been noted by Kleine in the intestine. Miss Robertson finds that the trypanosomes infect the salivary glands from the gut via the proboscis and the salivary ducts.



FIG. 97.—DEVELOPMENT OF *T. castellani* BRUCE 1903

1. In the vertebrate blood 2 and 3 in the mid-gut 4 and 5 in the hind-gut 6 in the salivary glands of the tsetse fly

(After Bruce Hamerton Bateman and Mackie)

Kinghorn and Yorke have shown that the salivary glands of *Glossina morsitans* become infected in a somewhat similar manner with *T. rhodesiense*.

Chagas has seen trypanosomes in the body cavity and salivary glands of *Lanus megistus* which are without doubt the forms

markedly influenced by the temperature of the air 75 to 85 °F being more favourable than 60° to 70° F and under favourable conditions the first stage of development can take place but not the later stages and flies may remain with the parasites incompletely



or general oedema and disease of the nervous system ; Secondary bacterial affection

MORBID ANATOMIC inflammation and œdema and dropsy which in human encephalitis as will be described later

INOCULATIONS —The parasites can be spread from one animal and from one species to another by inoculation of infected blood

AGGLUTININS —While in the body of the host agglutinins are formed for if blood containing trypanosomes is treated with the serum of an animal which has had one or more injections of blood containing the same parasite a rapid massing of the parasites into rosettes with the anterior ends pointing inwards and the flagella outwards takes place

This is called agglomeration and may last a few minutes the

precipitins

others

Chalmers and O Farrell have shown that *T. castellans* can be separated from *T. rhodesiense* by immune serum reactions *in vitro* and *in vivo*

IN  
may  
drugs

been noted by Chalmers and O Farrell

TOXINS —Uhlenhuth Hubener and Worthe have demonstrated the presence of endotoxins in *T. equiperdum* which observation supports McNeal's suggestion as to their presence and also the work of Martin Darre and Leber Free toxins do not exist but endotoxins can be set free by trypanolysis

Local Reservoirs —The long continued infectivity of *Glossina palpalis* after the removal of man from a district points to either long duration of infectivity in the fly or to a local reservoir which may perhaps be found in antelopes and perhaps other animals in the case of *T. castellans*

Cultivation —McNeal and Novy cultivated *T. lewisi* in the water of condensation of blood agar tubes in 1903 and obtained at 37° C



*Trypanosoma*

**Zoological Distribution**—It has been found in *Rana esculenta* Linnæus in *R. temporaria* Linnæus in Europe in *R. speciosa* in the Congo in *R. trinodis* in the Gambia. Whether the species found in *Hyla arborea* are truly *T. rotatorium* or not is uncertain.

**Morphology**—The pleomorphism exhibited by this form is so varied that it requires classification for as Chalachnikov has shown the following varieties exist:

1 *Flattened Forms*—(a) Simple plain forms (b) plain forms rolled on themselves (c) spiral forms

2 *Pectinated Forms*—(a) Pectinated spiral forms (b) cornucopial forms. In all these forms the undulating membrane is much folded and has a thickened edge. The flagellum which is short starts from the kinetocytoneucleus which is situated at a variable distance from the aflagellar extremity. The trophocytoneucleus is round or oval. The usual length varies from 40 to 80  $\mu$ , the breadth from 5 to 40  $\mu$  and the flagellum is about 10 to 12  $\mu$  long.

Doflein finds that in the blood and internal organs forms intermediate between the flagellate and the non flagellate organisms are found. The latter cannot divide.

**Life History**—The life history is but little known. Asexual multiplication takes place by

the trypanosome becoming round and losing its locomotor apparatus and dividing by mitosis. It is but rarely inoculable into other frogs but it grows in cultures especially upon Novy McNeal's medium but

**Cultivation**—It has been cultivated in acid bouillon blood media by

environment may become so altered under certain circumstances



FIG. 93—*Trypanosoma rotatorium* MAYER  
(After Dutton and Todd)



outlined by Noller while Minchin had he lived would probably have brought forward one

In the Trypanosominae the definitive and hence primitive host

elucidation but such forms as *Cystotrypanosoma*; *intestinale* Roubaud 1911 are worthy of more consideration and it is obvious that such forms deserve separate classifications and should form part of a tribe—*Cystotrypanae* with *Cystotrypanosoma* as type genus and *C. intestinale* as type species

As evolution proceeds so life histories tend to become complicated In this case the complication is the introduction of a vertebrate intermediary host and with the change in environment one meets with the large relatively slow moving trypanosome of the cold blooded vertebrate and the smaller quicker moving trypanosome of the warm blooded vertebrate They appear to us to deserve to be ranked into tribes and to merit more study The type of

A \* —  
1911—Tribe 1  
1

B. Live in a definitive invertebrate host and in a cold blooded intermediate vertebrate host —

Type genus —*Trypanosoma* Gruby 1843—Tribe 2 *Trypanosomae* Chalmers 1918

Type species —*T. rotatorium* Gruby 1843

C Live in a definitive invertebrate host and in a warm blooded intermediate vertebrate host —

Type genus *Castellanella* Chalmers 1918—Tribe 3 *Trypocastellanellae* Chalmers 1918

Co type species *C. gambiensis* (Dutton 1902)

*C. castellanii* (Kruse 1903)

In the present work we are chiefly concerned with the third tribe *Trypocastellanellae*

## SERIES A TRYPANOSOMES INFECTING INVERTEBRATA

### TRIBE 1 CYSTOTRYPANÆÆ

This tribe has not yet been fully studied but provisionally it can be classified as follows —





## SECTION I TRYPANOSOMES OF *Hydrineæ*

*Trypanosoma inopinatum* is believed to be a true parasite of the leech *Helobdella agira*, though according to some observers it is the same as the parasite of the frog

## SECTION II TRYPANOSOMES OF *Arachnida*

*T christophersi* Novy 1907 found in *Rhipicephalus sanguineus*, fed on dogs

## SECTION III TRYPANOSOMES OF *Hexapoda*

*Trypanosoma boyleyi* Lafont 1902 has been found in *Conorhinus rubrofasciatus* an insect which attacks man in Mauritius and Reunion

*Trypanosoma tullocki* Minchin 1907—This parasite closely resembles *C castellanis* from which it can be differentiated by the central round nucleus and the small centrosome It is found in *G palpalis*

Schaudinn 1904 also in *Culex pipiens* and further that the trypanosome found by Durham in *Stegomyia fasciata* which had been fed on bats should be looked upon as belonging to the mosquito

*T triatomæ* Kofoid and McCulloch 1916 is a parasite of *Triatoma protracta* found in nests of the wood rat *Neotoma fuscipes*

## SERIES B TRYPANOSOMES INFECTING COLD BLOODED VERTEBRATES

### TRIBE 2 TRYPANOSOMEÆ.

At present this tribe contains one genus—viz *Trypanosoma sensu stricto* as defined above and with *T rotatorium* Mayer, 1843 as the type

It is probable that as constituted the genus still contains a number of non defined genera but these require further investigation and we therefore divide the species into —

- Section I Trypanosomes of Fish
- Section II Trypanosomes of Amphibia
- Section III Trypanosomes of Reptilia

### SECTION I TRYPANOSOMES OF FISH

In 1841 the first known trypanosome was found by Valentin in the blood of *Salmo fario* the brown trout



and is thought to be spread by a leech—*Pontobdella muricata*; *T. sacco-*  
*branchi*: Castellani and Willey, 1905 in *Saccobranchnus fossilis* in the Lake of  
 Colombo, Ceylon.

## SECTION II : TRYPANOSOMES OF AMPHIBIA.

The trypanosomes of frogs were discovered by Gluge as far back as 1842  
 in the form of the largest trypanosome known—*i. e.*, *T. rotatorium*  
 It seems probable that leeches are the carriers of these parasites



FIG 102 — *Trypanosoma pertense* ROBERTSON  
 FIG 103 — *Trypanosoma vittata* ROBERTSON SHOWS THE TRY-  
 PANOSOME ROLLING UP PRIOR TO  
 DIVISION

(From drawing by Miss Robertson)

*Trypanosoma inopinatum* Ed and Et Sergent, 1904.

Synonyms.—*T. elegans* Franca and Athias, *T. undulans* Franca, *T. henderson*  
 Patton.

*Trypanosoma nelspruitense* Laveran, 1904

Discovered by Theiler in *R. angolensis* Bocage and in *R. theileri* Macquart.

*Trypanosoma somalense* Brumpt, 1906

In *Bufo reticulatus* from Somaliland.



FIG 91 — *Trypanosoma leschenaultii* ROBERTSON

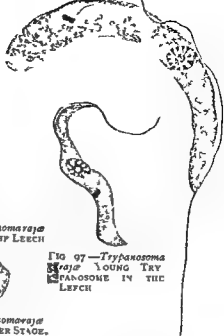


FIG 95 — *Trypanosoma raja*  
ROUND FORM IN THE LEECH

FIG 97 — *Trypanosoma raja* YOUNG TRY-  
PANOSOME IN THE  
LEECH



FIG 96 — *Trypanosoma raja*  
ROUND FORM OLDER STAGE.

FIG 99 — *Trypanosoma raja* POSSIBLY A  
FEMALE FORM IN  
THE LEECH



FIG 99 — *Trypano-  
soma raja* POSSI-  
BLY A MALE FORM  
IN THE LEECH

FIG 100 — *Trypanosoma raja* SLENDER FORM  
FROM THE PROBOSCIS  
OF THE LEECH

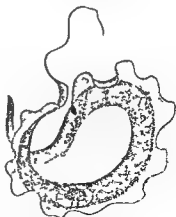


FIG 101 — *Trypanosoma raja*  
FULLY DEVELOPED TRYPA-  
NOSOME IN THE SKATE

(All after Miss Robertson)

- B Without schizogony in the vertebrate host —  
 I Enters red blood corpuscles—*Endotrypanum* Mesnil  
 and Brumont 1908  
 II Does not enter red blood corpuscles  
 (a)

Chalmers 1918

- (b) Final stage of development in the definitive host  
 in the salivary glands proboscis or hypo-  
 pharynx Infection inoculative  
 1 Polymorphic with granular cytoplasm small  
 kinetocell and well-developed undulating  
 membrane Final stage in the definitive  
 host takes place in the salivary glands—  
*Castellanella* Chalmers 1918  
 2 Monomorphic with non granular cytoplasm  
 large kinetocell and with or without  
 well developed undulating membrane  
 Final stage of development anterior but  
 not in the salivary glands—*Duttonella*  
 Chalmers 1918

The type species of these genera are as follows —

ca  
 1903)

*Duttonella vivax* synonym *Trypanosoma vivax* Ziemann 1903

## SECTION I TRYPANOSOMES OF AVES

distinct type

A few examples may be mentioned — *T. avium* Laveran 1903 in *Syrnium*  
*aluco* L. the wood-owl *T. confusum* Lube 1906 in *Agelaius phoeniceus* L.  
 and other North American birds *T. laverani* Novy and McNeal 1905 in  
 Mesnil 1904 in *Fed.*

*ictus senegalensis* and *T.*

## SECTION II THE TRYPANOSOMES OF THE MAMMALIA

Mammalian trypanosomes may be classified according to Laveran  
 and Mesnil into —

*Trypanosoma* sp (?)

Found by Tobey in the American newt *Dienycheilus viridescens*

## SECTION III TRYPANOSOMES OF REPTILIA

*Naja nigricollis*

## SERIES C TRYPANOSOMES INFECTING WARM BLOODED ANIMALS

## TRIBE 3. TRYPOCASTELLANELLEÆ

This tribe may be divided into —

SECTION I TRYPANOSOMES OF AVES — These are but little known and pending further study we must retain the old genus *Trypanosoma sensu lato*

SECTION II TRYPANOSOMES OF MAMMALIA — They may be classified as follows —

Series (a) — Non Pathogenic

1 *Classifiable* —

Genus *Lewissonella*

Genus *Endotrypanum*

2 *Unclassifiable* —

Old genus *Trypanosoma sensu lato* pending further work

Series (b) — Pathogenic

1 *Classifiable* —

Genus *Schizotrypanum*

Genus *Castellanella*

Genus *Duttonella*

2 *Unclassifiable* —

Old genus *Trypanosoma sensu lato* pending more research

3 *Little known*

The classifiable mammalian trypanosomes may be recognized by the characters given in the following table —

A With schizogony in the vertebrate host — *Schizotrypanum*  
Chagas 1909

mention as occurring between the kinetonucleus and the trophonucleus appears to have been also seen by Prowazek. Asexual reproduction may be

*Pneumocystis carinii*

*In the Invertebrate*—The rat flea *Ceratophyllus fasciatus* is the true carrier of *L. lewisi* as was first demonstrated by Nuttall and the rare development which may take place in louse *Polyplax spinulosa* is more of the nature of a natural culture than a proper development. The development in the flea has been studied by Swellengrebel and Strickland and more recently by Minchin and Thompson.

who finds that the micro- and macro-gametocytes undergo first a reduction of the trophonucleus from sixteen chromosomes to four and that then the microgametocyte gives rise to only one microgamete which fuses with the macrogamete forming an ookinete. This becomes a trypanosome by the

not to be compared with that in the flea.

*Cultivation*—Cultural experiments have been carried out successfully by Novy and McNeal on a medium prepared by mixing agar and defibrinated

Series (a) —The non pathogenic mammalian trypanosomes

Series (b) —The pathogenic mammalian trypanosomes

This classification is in our opinion in accord with evolutionary knowledge

## SERIES A THE NON PATHOGENIC MAMMALIAN TRYPANOSOMES

It is quite beyond the limits of this book to give detailed accounts of these trypanosomes and all that we can do is to attach a list of the more common with an account of a few

### Classifiable

Two genera are known—viz —

A Endoglobular forms known—*Endotrypanum*

B Endoglobular forms unknown—*Lewissonella*

### Unclassifiable

C Little known forms—*Trypanosoma sensu lato*

Genus *Lewissonella* Chalmers 1918

Redescription: —

Greater detail

*Lewissonella lewisi* (Saville Kent 1880)

Synonyms: — *T. lewisi* (Saville Kent 1880)

Remarks: — *T. lewisi* is a flagellate

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nut. eos

Life-History—*In the Vertebrate*—The life history in the rat has been worked out by Eremal and Hindle (Fig 8) p 384) The filament they



*Trypanosomes of Insectivora*—*T talpæ* Nabarro, *T soricis* Hadwen

*Trypanosomes of Fidentata*—*T legeri* Mesnil and Brimont 1910

*Trypanosomes of Carnivora*—*T pestanae* Bettencourt and França, 1905

*Trypanosoma duttoni* Throux 1905

Trypanosomes were found in fleas caught on infected animals but they did not show any development

*Trypanosoma musculi* Kendall 1906

This parasite was found in 8 per cent of the mice examined by Kendall in Panama. It was non pathogenic and resembled *T duttoni*.

*Trypanosoma microti* Laveran and Pettit 1909

Found in *Microtus arvalis* Pallas. It is 25 to 30  $\mu$  by 1.5  $\mu$ .

*Trypanosoma blanchardi* Brumpt 1905

In *Myoxis glis* the common dormouse. Like *T lewisi* but not inoculable into rats.

*Trypanosoma myoxi* R. Blanchard 1906

Found by Galli Valerio in *Muscardium (Myoxis) avellanarius* L., but nothing much is known about the parasite.

*Trypanosoma arvicanthidis* Delanoe 1915 from species of *Arvicanthus* and *T eburnense* Delanoe 1915 from *Musconcha* are varieties of *T lewisi*.

*Trypanosoma ericeti* Luhe 1906

but *Ceratophyllus fasciatus* Bosc is common

*Trypanosoma eunleui* R. Blanchard 1906

*Trypanosoma bandicotti* Langard 1904

This trypanosome is probably not the same as *T lewisi* which it resembles in being pathogenic to guinea pigs. It is found in *Nesokia bandicotti* Bechst in Bombay and the Deccan.

these changes

#### Genus *Endotrypanum* Mesnil and Brimont 1908

**Definition.**—Trypocastellapellæ found in mammals non pathogenic and with endoglobular forms

**Type Species**—*Endotrypanum schaudinni* Mesnil and Brimont 1908

*Endotrypanum schaudinni* Mesnil and Brimont 1908

#### Unclassifiable

The following is a list of the unclassified species divided according to the classification of the host and arranged according to known importance—

*Trypanosomes found in Monkeys*—America *T. minasense* Chagas 1909 *T. prowazeki* Gossler 1908 Asia *T. rheu* Terry 1911,

Mendall 1906 *T. grossi* Laveran and Pettit 1909 *T. microti* Laveran and Pettit 1909 *T. blanchardi* Brumpt 1905 (= *T. mivona* Blanchard) *T. eosomys* Hildwen *T. peromysci* Watson *T. criceti* Luhe 1906 *T. cuniculi* R. Blanchard 1906 *T. hindicolti* Lingard 1904 *T. nabiasi* Railliet *T. leporis sylvaticus* Watson *T. acouchi* Brimont *T. indicum* Luhe *T. citelli* Watson *T. spermophilis* Laveran *T. otospermophilis* Wellman and Wherry *T. tetrodonis* Bruce 1915

*Trypanosomes of Bats*—*T. vespertilionis* Battaglia 1904 *T. mega derma* Wenyon 1908 *T. mcolleorum* Ed and Et Sargent 1905 *T. limeatus* Iturbe and Gonzalez 1916





cultivated, and appears to develop in certain flies—for example, *Tabanus tropicus*, *T. lineola*, *Stomoxys calcitrans*, *S. geniculatus*, in the stomach of the last of which it has been found. Certainly it can be transmitted to healthy animals by the bites of flies and fleas. It is said that it can also be contracted by eating infected meat.

*T. striatus* Fabricius according to Mitzman can mechanically transmit surra.

*Castellanella Brucei* Plimmer and Bradford, 1889

Synonyms.—Perhaps *T. equi* Blacklock and Yorke, 1913. According to

*tans*) disseminated the disease.

The parasite is widespread throughout Africa, especially in Zululand, Northern Transvaal and its surrounding countries, also from Pretoria to Lake Nyassa in the basin of the Limpopo, in the basin of the Zambesi, in East Africa, where it causes nagana or the fly disease, and in Uganda where it is called 'jinja'.

**Morphology.**—The appearance of the parasite is worm-like, being 28 to 33  $\mu$  in length in horses and donkeys. The length is constant for the given animal but varies in different hosts, being 26 to 27  $\mu$  in rats, mice, guinea pigs, rabbits, and dogs. The anterior end (non-flagellate) is a truncated cone behind which lies the kinetonucleus as a well-marked rounded mass, posterior to which the flagellum arises. The trophonucleus lies in the middle of the body, and

developed undulating membrane. Movements active. Final stage in the definitive host is confined to the proboscis and hypopharynx.

**Type Species**—Bruce's Uganda strain of *T. ax* which is probably the same as *cazalboui*.

**Other Species**—The other species may be recognized as follows—

- (a) Kinetonucleus large and terminal. Undulating membrane well developed and simple. Invertebrate host a glossina—
- 1 Rats refractory—*Uniformis*
  - 2 Rats susceptible—*Itax*
  - 3 Only equidae and ruminants susceptible—*Caprae*
- (b) Kinetonucleus prominent and subterminal. Undulating membrane poorly developed—
- 1 Small 8-18 microns found in cattle—*Pecorum*
  - 2 Larger 14-24 microns found in monkeys—*Simiae*

B " " " "

Group 2 No part of the flagellum free

Group 3 Part of the flagellum may or may not be free

Group 4 Little known forms

## TRYPANOSOMES OF ANIMALS

### FORMS CLASSIFIABLE

#### Genus *Castellanella* Chalmers 1918

*Castellanella evansi* Steel 1885

**Synonyms**—*Spirocheta evansi* Steel 1885 *Hamatomonas evansi* Crookshank 1886 and *Psychomonas evansi* Crookshank 1896. According to Yorke and Blacklock *T. soudanense* and *T. venezuelense* according to Bruce *T. soudanense* Laveran

characterized by circular œdematous areas about the size of a two-shilling

synovial engorgement of the joints and tendon sheaths and enlargement of the lymphatic glands particularly the inguinal. The temperature is often raised to 39° C (102.2° F) in the evening and falls to 38.5° C (101.4° F) in the morning.

*Stage 3 or the Period of Anæmia and Paralysis*—The animal now becomes very anæmic with pale mucosæ and emaciation is marked. There are often superficial abscesses which do not heal and some conjunctivitis and ulcerative keratitis. Micturition is difficult and the urine is thick. Sensibility is

becomes affected. Other lesions are gelatinous exudation under the skin, serous effusions into the pleural and peritoneal cavities, wasting and pallor of muscles with fatty degeneration and an interstitial keratitis.

### Genus *Duttonella* Chalmers 1918

*Duttonella vivax* Ziemann 1905

It has a karyonucleus and micronucleus and has its flagella at the anterior end. The flagella are attached to the proboscis.

*Duttonella capræ* Kleine 1910

It is a heavily built trypanosome with very rapid movements measuring

**Cultivation.**—Novy and McNeal have cultivated *C. brucei* in the same manner as *Lewissonella lewisi* and found some evidence of a toxin but it only grows exceptionally in the water of condensation from the agar medium which contains half or less than half its volume of blood. Agglomeration takes place under various circumstances—e.g. mixture with immune blood or a few drops of dilute acetic acid etc.

**Pathogenicity.**—The disease can therefore be spread by the bites of certain tsetse flies particularly *G. morsitans* and perhaps the others mentioned above.

It can however be also spread by inoculation and by eating the blood of animals recently dead from the disease. The incubation period is about ten days and the effects produced in animals vary considerably in the following manner—

1. It is an acute disease in mice rats dogs monkeys cats etc. dogs dying in two to six days rats in three to six days

*Castellanella equiperdum* Doflein 1901

Synonym—*T. rougei* Laveran and Mesnil

ho

in



- C *Invertebrate host a Glossina* —  
Rats refractory Large forms 24 microns—*Cazalbois*
- D *Invertebrate host unknown* —
- I Attacks horses —
- (a) In Venezuela — *Venezuelense*
- (b) In Morocco separated by cross immunity — *Maroccanus*
- (c) In Algeria separated by cross immunity — *Berberum*
- II Attacks horses and cattle —  
In Annam—*Annamense*
- III Attacks cattle —  
In Italian Somaliland—*Cellis*
- *Trypanosoma equinum* Vosges 1901

“

•

the pleura the pericardium and the spinal anal

***Trypanosoma soudanense* Laveran 1907**

Type of *Trypanosoma evansi* causing tahaga in dromedaries in the Upper Niger el debab in Southern Algeria and the zoufana in horses in Southern Algeria Carriers Tabanidæ Yorke and Blacklock consider this to be the same as *Castellanella evansi*

***Trypanosoma togolense* Mesnil and Brimont 1909**

Type of *Castellanella etansi* parasitic in horses and cattle and the cause of nagana in Togoland



## GROUP 2 NO PART OF THE FLAGELLUM FREE

*Trypanosoma congolense* Broden 1904

*Trypanosoma congolense* Broden 1904

infective trypanosomes are in the hypopharynx

*Trypanosoma dimorphon* Laveran and Mesnil 1904

This trypanosome was discovered by Dutton and Todd in 1904 in horses on the Gambia and is now known to exist in several parts of Africa where perhaps a wide equatorial belt across the continent is affected. It is also found in cattle dogs pigs sheep and goats

**Morphology**—It exists in three forms

1 *Tadpole Form*—Found in the early stage of the disease 11 to 13  $\mu$  long and 6 to 8  $\mu$  broad



FIG 104 —*Trypanosoma dimorphon* LAVERAN AND MESNIL  
(After Dutton and Todd)

2 *Stumpy Form*—Seen when the disease is not too far advanced and

**Trypanosoma (Duttonella) cazalbei Laveran, 1906**

This organism, which should be placed in the genus *Duttonella* by Cazalbei in 1904 in "souma," Nigeria. The disease is known in Congo, the Congo and Rhodesia donkeys or chronic months situate a near the folded, as the usual

**Trypanosoma hippileum Darling 1910**

This trypanosome causes a disease called murrina among mules and was first described in some animals imported from the to Panama in length with The kinetocoe is a character much folded

kidney liver, e  
siderable amou

**Trypanosoma venezuelense Mesnil 1910**

Type of *Castellanella evansi*, and considered by Yorke and Blacklock to be identical. Attacks horses in Venezuela. carrier unknown

**Trypanosoma annamense Laveran 1911**

Type of *Castellanella evansi* causes disease in horses and cattle in Annam. Carriers: Tabanidae and Hippoboscidae

Trypanosoma

When a classification is desired it is always necessary to attempt to discover the characters of the original species, which in this case is *T. gambiense* Dutton 1902. Sixteen years have passed since the slides containing the original forms were made, and, the only method of comparison is now

comparison by measuring one thousand non dividing forms in the original slides. As far as measurements go these strains are very similar but as we have repeatedly insisted morphology often may not help in separating closely related but perhaps quite distinct species which require to be studied serologically and with regard to animal pathogenicity and in cases of human infection with regard to the nature of the disease in man. Thus Stephens has pointed out that *T. lewisi* and *T. rabinowitschi*, *T. brucei* and *T. evansi*, *T. pecaudi* and *T. ugandæ*, *T. rhodesiense* and *T. pecaudi* are indistinguishable

in experiments, as well as by morphological characters, and we have suggested for years that the name *T. gambiense* covered a number of different forms which at the present time is generally admitted with regard to *T. rhodesiense*. And why not? Are there not a number of different trypanosomes in wild animals in Africa and is it impossible that man should from time to time become infected by one of these even if it does not appear in epidemic form in the human race? To exemplify we draw attention to an organism resembling *T. vivax* found by Macfie in man.

Sir David Bruce believes that the organism which he described in 1910, is the same as *T. brucei*. He can hardly be so because La Motte immunized a ram against *T. brucei*, and then infected it with *T. rhodesiense* an acutely lethal infection ensuing. The serological experiments of Chalmers and O Farrell *in vitro* and *in vivo* also show the same marked differences between *T. rhodesiense* and another posterior nucleate trypanosome. These experiments to our mind

**Pathogenicity**—The symptoms in the horse begin with loss of vigour followed in two to three weeks by fever. During the next month the weakness is more marked and the abdomen swells, the testicles hang down and are oedematous, the coat becomes staring, the animal looks apathetic and death

**Trypanosoma frobeniusi** Weissenborn 1911

Allied to *T. dimorphon* and *T. congolense* and found in horses in Togoland

**Trypanosoma nanum** Laveran 1905

Balfour in 1904 discovered this parasite in cattle which appeared to be ill

GROUP 3 PART OF THE FLAGELLUM MAY OR MAY NOT BE FREE

**Trypanosoma pecandi** Laveran 1907

In the French Sudan in addition to m. boris and souma there is a third disease—baleri in Equidae—caused by *T. pecandi* with two forms like *T. dimorphon*—(1) long and slender (25 to 35  $\mu$  by 1.5  $\mu$ ) (2) short and broad (14 to 20  $\mu$  by 3 to 4  $\mu$ ) Carriers *Glossina longipalpis* rarely *G. palpalis*

LITTLE KNOWN TRYPANOSOMES

*Trypanosoma elephantinum* ...

...

...

... similar to those found in surra

*Trypanosoma bovis* Kleme was found in sick cattle near Tanganyika

THE TRYPANOSOMES OF MAN

There is evidence that man is infected with a variety of trypanosomes, the number of which is likely to be increased in the near future. Those described in man are—

the same organism as that described by Scott Macfie in 1913 as *T. nigeriense* Yorke and Blacklock in 1915 consider that man is the chief reservoir of this parasite in Sierra Leone where sleeping



FIG 105—*Castellanella gambiensis* (DUTTON 1902) ( $\times 1,000$  DIAMETERS)  
Long form from the original Gambia fever case (Mr K) discovered by Dutton (Photomicrograph)

sickness is very chronic and difficult to recognize. A secondary reservoir is in cattle. They record two cases with very mild symptoms: Sartory Lasseur and Brisaud record *C. gambiensis*



FIG 106—*Castellanella gambiensis* (DUTTON 1902) ( $\times 1,200$  DIAMETERS)  
Original Gambia specimens from a rat inoculated from the second case of Gambia fever showing polymorphism—i.e. long intermediate and short forms

trypanosomiasis in a French soldier who had left Africa for eight years and had never been in a tsetse fly area. In Africa he lived

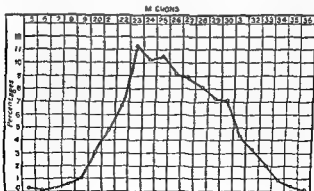


FIG 107—*Castellanella gambiensis* (DUTTON) . CHART OF LENGTHS

in Algiers at El Golea and went to Timmoun. Bagshawe draws attention to Neveu's 1888 and 1890 observations which have never been explained.

times from *T. brucei* but its altered environment in man has changed its physiological characters. As the fly remains the same one would expect this portion of the life cycle to be similar in the two trypanosomes.

We look upon *T. nigriense* and *T. gambiense* var. *longum* as belonging to *T. brucei*, and as separated from the normal fly by

parasites of man —

A Belonging to the genus *Castellanella* —

1 *Castellanella gambiensis* (Dutton 1902)

2 *Castellanella castellani* (Krusc 1903)

3 *Castellanella rhodesiensis* (Stephens and Grantham 1910)

B Belonging to the genus *Duttonella* —

4 *Duttonella vivax* (Ziemann 1903) var. *Macfiensis*  
(Castellani and Chalmers 1918)

C Belonging to the genus *Schizotrypanum* —

5 *Schizotrypanum cruzi* Chagas 1909

These five species may be differentiated as follows —

A With schizogony—*S. cruzi*

B Without schizogony —

I Monomorphic—*D. vivax*

II Polymorphic —

(a) Posteriorly nucleate—*C. rhodesiensis*

(b) Not posteriorly nucleate —

1 Animal infections chronic and comparatively mild. Common North West Africa—*C. gambiensis*

2 Animal infections severe. Common Equatorial Africa—*C. castellani*

*Castellanella gambiensis* (Dutton 1902)

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va		1050
		tro

never thought by them to be connected with sleeping sickness. The organism had however been previously seen in the blood of man and imperfectly described by Nepveu. We consider it to be



enlarged lymphatic glands by scarification of the eruption or by puncture of the skin

Salvin Moore an cycle in the rat in to a maximum in 14 to 21 at which the then increase again in numbers and reappear in the peripheral blood Their investigations give the following results The parasite may

coil on itself but eventually is connected with the trophonucleus

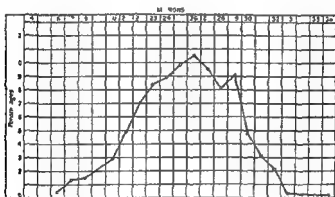


FIG 109.—*Castellanella castellanus* CHART OF LENGTHS

The trypanosomes now decrease in numbers in the peripheral blood and are found in the lungs spleen and bone marrow In these organs the protoplasm becomes detached from the periphery of the nucleus which lies in a clear space The nucleus contracts and a large clear vesicle forms in connection with it and around both a cytoplasmic sheath is formed The rest of the cell body now disintegrates and the flagellum with the kinetocore may be seen lying detached These bodies now become lodged in the

latent blood culture consist of a flattened nucleus containing a centrosome and attached to a vesicle the whole being surrounded by a ring of cytoplasm This latent phase has been confirmed by Pantham Just before the reappearance of the trypanosomes in the per-

**Morphology.**—It is a polymorphic trypanosome and morphologically does not differ essentially from *C. castellanii* of which a detailed description is given below.

**Life-History.**—This has not been fully studied and it is not definitely known whether it takes place in *Glossina palpalis* or in

symptoms are often less severe though the termination is fatal without treatment, to which it appears to be more amenable.

### *Castellanella castellanii* (Kruse 1903)

Tr. 1 12

the cause of the disease in 1903, he asserted also the probable plurality of species of the trypanosomes affecting man, in analogy to what takes place in the leishmaniasis.

that the transmission is not merely mechanical but also takes place after a period during which the fly is non-infective, the deduction being

**Morphology.**—*C. Castellani* measures from 14 to 22  $\mu$  in length and from 2 to 3  $\mu$  in width.

granules in the cytoplasm which at the posterior end runs along the flagellum for a considerable distance.

**Asexual Reproduction.**—The life-history of the parasite in the human body is not accurately known. It can often be obtained from the peripheral blood but sometimes the most prolonged search fails to demonstrate it. It can then be found by puncturing the



FIG. 108 — *Castellanella castellanii* (Kruse 1903)

The original trypanosome found by Castellani in the cerebro-spinal fluid of

is often seen situated about the periphery of the cell and may be some chromidia.

sequent casting off of the aflagellar end with the remains of the flagellum. The result of this posterior and anterior reduction in length is to produce a rounded body with a trophonucleus and a kinetonucleus which surrounds itself with a capsule and forms the latent body or the *post flagellate stage* of the life cycle. These cysts are about 3 to 4  $\mu$  in diameter.

After a time these bodies become the *preflagellate stage*, increase in size and length and eventually a flagellum grows out from the kinetonucleus and gives rise to the undulating membrane and thus again forms the usual *flagellate stage*.

(b) *In the Invertebrate*—*Glossina morsitans* when fed upon infected animals is capable of spreading the infection mechanically for about twenty four hours after which period it ceases to be infective and remains non infective for at least fourteen days after which about 5 per cent of flies become again infective when trypanosomes can be demonstrated in its alimentary canal and in its salivary glands.



FIG. 112—*Castellaniella rhodesiensis* (STEPHENS AND FANTHAM)  
(After Stephens and Fantham)

1 Long narrow form      4 nucleus passing to aflagellar end      5 nucleus at the aflagellar end

Γ

(a) *Sero Diagnosis*—(1) Attachment experiments are very inconstant and do not distinguish between the two.

(2) Trypanolysis does not help being also inconstant.

(3) *C. castellani* is resistant to human serum. *C. rhodesiensis* is less resistant.

(b) *Crossed Immunity*—An animal having an immunity against *C. castellani* can be infected by *C. rhodesiensis*. The reverse experiment has not yet been conducted.

We may therefore conclude that *C. castellani* and *C. rhodesiensis* are different species.

Is *C. rhodesiensis* a variety of *C. brucei*? The answer is No because Laveran has shown that animals immunized against



FIG. 110.—*Castellaniella castellanii* (KRUZE, 1901)

(Forms found in the cerebro-spinal fluid)

Brumpt finds that the parasite lives well in *Clinocoris lectularius*, *C. rotundatus*, *Clinocoris boueti* and *Ornithodoros moubata*. His account of the cycle of development is as follows—Starting with the trypanosome in the posterior part of the intestine when this is inoculated into the vertebrate it enters the cells of the body and becomes Leishmania like bodies which eventually develop into free-swimming trypanosomes from which the form capable of continuing the infection in the vertebrate or invertebrate is produced. In the invertebrate these become crithidia like forms and

the fæces  
vitiiceps  
Rhipi

*cephalus sanguineus*

Culture.—*S. cruzi* is easily cultivated upon the Novy McNeal

larvæ of *Lanus* cease to be infective to vertebrates and first become so on the eighth to the tenth day after which they remain infective for a long period. The parasites so introduced into man give rise to American trypanosomiasis.

Reduction in Virulence.—*S. cruzi* when repeatedly passed through animals of the same species become weakened in virulence but regain this when transmitted to a fresh species.

Infectivity.—The infected monkey is infective for the bug while the infected guinea pig is not.

#### *Duttonella vivax* Ziemann 1905 var *macfieensis*

Synonym.—*Trypanosoma vivax* (Ziemann 1905) *pro parte*

In 1917 Macfie described a monomorphic trypanosome very closely resembling *T. vivax* but slightly smaller with the crest in

flagellum

*Castellaneella nigertensis* Macfie 1913

— on the

*C. brucei* are susceptible to *C. rhodesiensis*. Is *C. rhodesiensis* a variety of *T. pecaudi*? No because the former is more virulent than the latter to animals and because sleeping sickness is unknown in the region where malaria is intense and finally because an animal immunized against *C. rhodesiensis* is not immune against *T. pecaudi*.

We may therefore conclude that *C. rhodesiensis* Stephens and Fantham 1910 is a good species.

**Cultures**—Thomson has cultured it with partial success on a modification of the Novy McNeal Nicolle medium.

**Vertebrate Reservoir**—It is claimed that the larger game animals are the reservoir of this trypanosome.

**Pathogenicity**.—*C. rhodesiensis* is the cause of one form of sleeping sickness.

### Schizotrypanum cruzi Chagas 1905

**Synonym**—*Trypanosoma cruzi* Chagas 1905

**History**—This trypanosome was discovered by Chagas in the intestine of *Lamys megistus* Burmeister in Brazil and later it was found in the blood of a child suffering from irregular fever, progressive anemia and enlargement of various groups of lymphatic



FIG. 123.—SCHIZOGONY OF *Schizotrypanum cruzi* CHAGAS  
(After Chagas.)

1 merozoite in red blood cell 2 parasite totally enclosed in red cell in flagellum or undulating membrane 3 5 parasites partially enclosed in red cell 6 7 parasites in human blood 8 11 parasites in the lungs of *Callithrix* 12 13 initial forms of schizogony 14 15 schizogony in the lungs of *Callithrix*

glands. The trypanosome was characterized by the presence of a large kinetoplast and by the facility with which it could be cultivated on blood agar. In 1910 it Chagas published a series of papers upon the life history of the parasite and the symptomatology of the disease which it produces. In 1911 Vinnia studied the pathological anatomy while further studies on the parasite were made by Brumpt, Martin, Mayer, Pocha, Lima, and others.

**Morphology**—In the peripheral blood of man *S. cruzi* appears in two forms—either free or in the red blood corpuscles.

... ..

*Leucocytozoon danilewskyi* Ziemann, 1898

Synonyms.—*Hamamæba ziemanni* Laveran 1902, *Spirochæta ziemanni* Schaudinn, 1904, *Plasmodium ziemanni* Blanchard, 1905 *Leucocytozoon*



FIG 118—INTRACELLULAR FORM OF *Leucocytozoon loati*  
(After Sambon)



FIG 119—INTRACELLULAR MICROGAMETOCYTE OF *Leucocytozoon loati*  
(After Seligmann and Sambon)



FIG 120—INTRACELLULAR MACROGAMETOCYTE OF *Leucocytozoon loati*  
(After Seligmann and Sambon)

as the original *C. ginsiensis* of which it appears to be merely a variant. We have placed it as a synonym of *C. ginsiensis*.

**Castellanella lanfranchii** Lanfranchi 1915

It approximates to *C. evansi* and is the organism with which Lanfranchi accidentally inoculated himself. As regards precipitating and complement fixation power is very similar to *C. evansi* but as regards trypanolytic action of the serum it approximates more *C. castellani*.

APPENDIX INCERTÆ SEDIS

In the ... ..

by Lanham. They are only definitely known to occur in birds and must be distinguished from the Hemogregarines of mammals.

**Leucocytozoon** Danilewsky 1889

Synonyms — *Hamamada* Laveran 1903 *Spirochala* Schaudinn 1904  
*Trypanomorpha* Woodcock 1906

The leucocytozoa were first described by Danilewsky between 1884 and 1886 in the blood of the wood owl (*Syrnium aluco*) and other Strigidae.



FIG. 117.—*Leucocytozoon danilewsky* ZIEGLER  
(After Schaudinn)

From the left to the right: a macrogametocyte free; same attached to a blood cell; same enclosed in a blood cell; a free microgametocyte; same enclosed in a blood cell.

He described ...



**Microgamete**—The microgametocyte escapes from the capsule like periplast and its nucleus breaks up into eight double chromosomes which are reduced to eight single chromosomes. These travel to the periphery and

*proteus*

Fig. 1. 2. 3. 4. 5. 6. 7. 8. 9. 10. 11. 12. 13. 14. 15. 16. 17. 18. 19. 20. 21. 22. 23. 24. 25. 26. 27. 28. 29. 30. 31. 32. 33. 34. 35. 36. 37. 38. 39. 40. 41. 42. 43. 44. 45. 46. 47. 48. 49. 50. 51. 52. 53. 54. 55. 56. 57. 58. 59. 60. 61. 62. 63. 64. 65. 66. 67. 68. 69. 70. 71. 72. 73. 74. 75. 76. 77. 78. 79. 80. 81. 82. 83. 84. 85. 86. 87. 88. 89. 90. 91. 92. 93. 94. 95. 96. 97. 98. 99. 100.

(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (11) (12) (13) (14) (15) (16) (17) (18) (19) (20) (21) (22) (23) (24) (25) (26) (27) (28) (29) (30) (31) (32) (33) (34) (35) (36) (37) (38) (39) (40) (41) (42) (43) (44) (45) (46) (47) (48) (49) (50) (51) (52) (53) (54) (55) (56) (57) (58) (59) (60) (61) (62) (63) (64) (65) (66) (67) (68) (69) (70) (71) (72) (73) (74) (75) (76) (77) (78) (79) (80) (81) (82) (83) (84) (85) (86) (87) (88) (89) (90) (91) (92) (93) (94) (95) (96) (97) (98) (99) (100)

These trypanosomes especially the male are very minute and reproduce by longitudinal division during which they do not separate at once but remain attached posteriorly. Couples attached to one another may extend

body enclosing a leucocytozoon

Ziemann Schaudinn Dutton Todd and Tobey believe that it is a portion of the parasite itself—the periplast—and that it may enclose a red blood cell

*pallidum*

Recent work has tended to confirm the doubts thrown on Schaudinn's work but the general appearance of a Leucocytozoon in its cell is sometimes remarkably similar to a trypanosome. It is possible that there may be

of the trypanosome



FIG 121—*Leucocytozoon danilewskyi* ZIEMANN MICROGAMETE  
(After Schaudinn)



FIG 122—*Leucocytozoon danilewskyi* ZIEMANN MACROGAMETE  
(After Schaudinn)



FIG 123—*Leucocytozoon danilewskyi* ZIEMANN THE DEVELOPMENT OF THE  
OOKINETE AND THE FORMATION OF SMALL TRYPANIFORM BODIES  
(After Schaudinn)

(a) *Endocellular Resting Stage*—In the intracellular stage the parasite is quite spindle-shaped the ends being composed of ectoplasm while the endoplasm forms a dark oval central mass containing the trophonucleus close to which the kinetonucleus is situated. The flagellum has disappeared but

in pairs

(2) *Resting Stage*—This differs from the microgametocyte in being large

d ham  
are sucked



different parasites confused under the term *Leucocytozoon* and their life histories may be different as Fantham's work presently to be described is opposed to Schaudinn's work

*Leucocytozoon lovati* Sambon and Sehemam 1907

*Spirochaetacea* Fantham 1908

Synonyms.—*Prostagellata* Doflein *Spiroschaudinidae* Sambon,  
 07. 52. "

The cytoplasm is divided into endoplasm and ectoplasm, and is surrounded by a flexible, chitinous periplast. The nucleus consists of a spiral achromatic filament, on which are arranged transverse bars and rodlets of deeply staining chromatin.

end in *C. anodontæ*, in the latter a short stiff process of periplast projects from it which is considered by some observers to be a flagellum

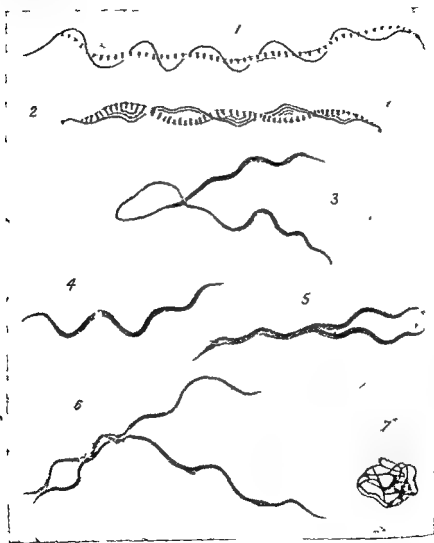


FIG 124—SPIROCHETES

— chromatic  
2 *C. balbi*  
3 7 Spiro-  
rasiology  
■ possible

points out that the strongest arguments in favour of their being bacteria are —

- 1 Diffuse character of nucleus somewhat like *Bacillus b. ischilus*
- 2 The possible occurrence of transverse fission
- 3 The absence of a typical kinetonucleus

While the points in favour of their belonging to the protozoa are —

- 1 The possession of an undulating membrane
- 2 The occurrence of longitudinal division (denied by Schellach)

related to the trypanosomes. Spirochætes are however distinctly peculiar particularly with regard to their diffuse nucleus but there appears to be almost a series from the diffuse nucleus of the bacteria through the achromatic spiral with its attached chromosomes of the spirochætes to the condensed nucleus of the higher protozoa. Schellach derives them phylogenetically from the oscillatory cyanophæes genus *Spirulina*.

**Morphology**—Spirochætes vary much in size from *Spiroschaudinnia recurrentis* which is 8  $\mu$  in length to *Cristispira balbiani* which may be 150  $\mu$  in length and 2 to 3  $\mu$  in thickness. The type species *Spirochæta plicatilis* was found in muddy water by Ehrenberg in 1833 and was said by Schaudinn in 1905 to possess an undulating membrane. Sambon however considers that this so

are here followed. These organisms are long wavy and thread-like composed of cytoplasm which can be differentiated into an ectoplasm which is generally converted into a thin flexible chitinous membrane the periplast. This is continued laterally into a spirally arranged membrane the crista containing longitudinally arranged fibrillæ and having a thickened border composed of chromatin. The longitudinal fibrillæ are composed of eight to nine principal and numerous secondary fibrillæ which are contractile, and are called the myoneme fibrillæ (Fig 124). The membrane or crista which does not markedly undulate helps in locomotion which is rapid and takes place by a wave-like flexion of the body

granules) at each end of the periplast in *C. balbiani* and only at one

a firm periplast with a very tenuous membrane which is often invisible. The nucleus consists of granules of chromatin distributed along the body.

giving rise to a number of round or oval granules probably the

same as the infective granules of Fry and Balfour which are known to escape from one end or the other of the periplastic sheath when in the internal organs. These infective granules enter the red cells and divide into a number of merozoites which escape from the red cell and enter the liquor sanguinis but their further development is unknown though it is possible that they become spirochætes.

Fantham's observations have confirmed this granule stage of the life-history but it must not be forgotten that every granule seen in a spirochæte is *not* an infective granule which our own observations support.

Mitchin regarded these granules as true endogenous chromidial buds and considered therefore that the term infective granule should be replaced by the term endogenous bud formation.



FIG 126.—LIFE CYCLE OF *Spiroschaus dunnii marchouxii* NUTTALL

(After Hindle from the *Journal of Parasitology*)

Reproduction takes place by both longitudinal and transverse

*Spirochaeta* Ehrenberg 1838 *sensu stricto*

*Cristispira* Gross 1910

*Saprosira* Gross 1911

*Pseudospira* Dobell 1912

*Spiroschaudinna* Sambon 1907

But it is only the last genus which contains the forms of importance in tropical medicine

Noguchi has created the genus *Lethospira*

the spiro  
has observed  
rod structure

*Cristispira balbianii* Certés 1882

This spirochaete is for the most part  
are apparently aff  
well defined undu  
diffuse nucleus of r  
been described

*Cristispira anodontae* Keysseltz 1906

Synonym — *Spirochaeta anodontae* Keysseltz 1906 *nomen nudum* Schellach 1909  
*S. anodontae* was found in the crystalline style of *Anodonta mutabilis* by  
Keysseltz in 1906 and in that of *A. cyanea* by Lanham in 1908



FIG 125—DIAGRAM OF *S. d. thos* SHOWING CHROMATIN GRANULES  
POINTED ENDS AND SLIGHT MEMBRANE EDGE (After Fantham)

It is 10 μm long

*Spiroschaudinna* Sambon 1907

Spirochaetidae parasitic in the blood and tissues of vertebrates  
and in some blood sucking invertebrates

Remarks — This genus as we believe would happen is now  
optimal though



the virulence diminishes. The subcultures do not lose their virulence even after the ninth passage.

He has also cultivated in this way *S. recurrentis* Lebert, 1874 when the maximum growth occurs on the seventh day, *S. rossi* Nuttall 1905 with a maximum on the ninth day and *S. novyi*, which is the most difficult with a maximum on the seventh day. *S. marchouvi* has also been cultivated. Bronfenbrenner in 1914 simplified this method of cultivation.

**Carriers**—The *Spiroschaudinnia* are spread by the agency of ticks and lice.

**Method of Infection**—The *Spiroschaudinnia* infect the ova of the tick and so pass into the second generation from which they escape in the fæces and enter the wounds made by the tick when it bites, and so infect the vertebrate host. Whether the same

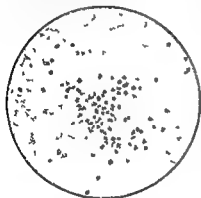


FIG 130—GRANULES IN AN INFECTED EGG

(From a microphotograph by Sir William Leishman)



FIG 131—MASSES OF GRANULES IN MALPIGHIAN TUBULE OF A YOUNG UNFED NYMPH ( $\times 8000$ )

(From a microphotograph by Sir William Leishman)

method of development takes place in the louse is not known but

own skin. The *Spiroschaudinnia*, escaping from the crushed louse enter the vertebrate host through the abrasions caused by the scratches. Both these are contaminative methods of infection. It is believed that *Spiroschaudinnia* in the vertebrate can pass from the mother via the placenta to the fœtus thus giving rise to an hereditary method of infection in contradistinction to the contaminative methods mentioned above.

## SPIROSCHAUDINIA

fully developed spirochaetes. Infection takes place towards the end of life by the excretion from the Malpighian tubules which contains the spirochaetes passing into the wound caused by the bite.

Some of the *Spiroschaudinia* on entering the tick pass into the alimentary canal and undergo multiple transverse division while they may live for some weeks in the gut.

**Cultivation**—Noguchi has successfully cultivated *S. dulcis* in sterile ascitic or hydrocele fluid to which a piece of fresh kidney

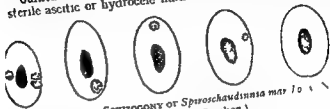


FIG 127—SCHIZOGONY OF *Spiroschaudinia* *maritima* (After Sambon)

has been added. For inoculation of this medium a few drops of the citrated heart blood from a mouse forty seventy two hours after infection

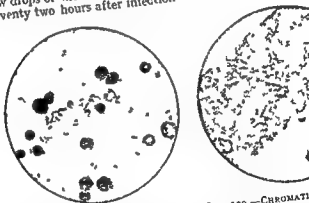


FIG 128—DEVELOPMENT OF *S. Dulcis* (X 1000)

The small dots are chromatin bodies and the large granules are digested blood while the pale outlines are spirochaetes in the contents of the intestinal sac of a tick two days after an infective feed (From a microphotograph by Sir William Leishman)

FIG 129—CHROMATIN BODIES IN THE MALPIGHIAN TUBE OF A TICK SIX DAYS AFTER FEED (X 1000)  
(From a microphotograph by Sir William Leishman)

At a temperature of 37° C the maximum growth is reached on the eighth to ninth day after which disintegration begins in total disappearance about the fifteenth day but is best made from the fourth to the ninth day but

length while the other is merely pointed. No undulating membrane could be differentiated but he states that he was able to demonstrate lateral cilia which he considers to be artificial but this in our opinion is highly suggestive of the presence of an undulating membrane. Coloured granules could be made out when stained by Giemsa. Reproduction was usually by transverse division.

**Life-History**—The spirochetes are found in the blood during the attacks of fever and equally distributed in the organs in the apyrexial interval.

**Inoculation**—It can be inoculated into monkeys but small rodents are especially susceptible. Subinoculations can be made from monkey to monkey and from mouse to mouse.

**Immunity**—Serum of animals immunized for *S. novyi* is without effect upon *S. recurrentis*, *S. duttoni* or *S. carteri*.

**Cultivation**—It has been cultivated by Noguchi as mentioned above (see p. 441).

**Pathogenicity**—It is the cause of North American relapsing fever.

#### *Spiroschaudinnia carteri* Manson 1907

*S. car*

thinner

immuni

and with difficulty into mice and can be subinoculated from monkey to monkey or mouse to mouse.

Novy and Knapp give the following differences between *S. carteri*, *S. duttoni* and *S. novyi*—

Character	<i>Spiroschaudinnia novyi</i>	<i>Spiroschaudinnia duttoni</i>	<i>Spiroschaudinnia carteri</i>
Length of simple cell	8 $\mu$	16 $\mu$	8 $\mu$
Length of double cell	16-20 $\mu$	30 $\mu$	16-20 $\mu$
Width	0.25 $\mu$	0.2 $\mu$	0.2 $\mu$
Number of turns in a single cell	2.3 $\mu$	2.5 $\mu$	2.3 $\mu$
Distance between the turns	1.5 $\mu$	4.5 $\mu$	2.3 $\mu$
Movement	Vigorous	Little	—
Number in peripheral blood	Many	Few	Many

According to Strong's experiments rats immunized against *S. recurrentis* and *S. novyi* are immune to *S. carteri*. He therefore believes these three strains to be closely allied if not identical.

They can also be distinguished by agglutination immunization tests by Pfeiffer's reaction and by certain animals being susceptible to some species and not to others. Mackie has suggested that *S. carteri* may be transmitted by a pediculus. It is the cause of Asiatic relapsing fever.

## I HUMAN SPIROCHÆTES

## A BLOOD SPIROCHÆTES

*Spiroschaudinnia recurrentis* Lebert 1874

Synonyms—*Spirochæte recurrentis* Lebert 1874 *S obermeyer* Cohn 1875

This spirochæte was discovered by Obermeyer in cases of relapsing fever in Berlin

**Morphology**—It exists in the blood in short and long forms. The short forms which are from 7 to 9  $\mu$  in length are probably early stages. The long forms 16 to 10  $\mu$  are probably late stages.

The short form which is considered to be one cell is two to three  $\mu$  in length.

The short form is said by Novy and Knapp to have a long flagellum at one end while the other has a faint appendage. The presence of flagella in this as well as in other spirochætes is denied by Nuttall.

**Life History**—This spirochæte is pathogenic to man, monkeys, rats and mice but these latter have to be infected from a monkey. Rabbits or guinea pigs are not susceptible. It is found in the peripheral blood during the attacks and elapses but not in the intermission unless occasionally after very protracted search.



FIG. 12.—*SPIROSCHAUDINIA* FROM A CASE OF A LATE RELAPSING FEVER (From a microphotograph by J. J. Bell)

It can be transmitted to man by the bed bug—succeeded on use to mouse. Lice have also been used as carriers. Most authorities consider lice to be

carriers  
**Immunity**—The organism is without pathogenicity.

440  
Italian and German workers The spirochaetes found in Japan and

where it is rare

The organism can enter through the alimentary canal via  
water

The spirochaetes are 6-9 microns long on an average but may

Guinea pi  
peritoneally  
illness but

to eight days and  
Rabbits cannot be  
re culture from the  
ving this on blood

#### **Spiroschaudinna hebdomadis Ido Ito and Wani 1918**

Morphologically similar to *S. icterohaemorrhagiae* differs serologically  
Found by Ido Ito and Wani in cases of a seven day fever called Nankayami  
which somewhat resembles atypical Weil's disease

The field mouse (*Miscotus montebelli*) seems to be the normal host of the  
spirochaete

#### **Spiroschaudinna in Yellow Fever**

### **B CUTANEOUS SPIROCHAETES**

#### **Spiroschaudinna vineenti Blanchard 1906**

*Spiroschaudinna vineenti* Prowazek 1907

to 20  $\mu$  in length with a well  
her short flagellum Male and  
wazek Division is longitudinal

**Spiroschaudinna berbera** Sergent and Toley, 1910

*Spiroschaudinna* with minimal length of 12  $\mu$  and irregular open spirals or flexures. It can be inoculated into monkeys (*Macacus cynocephalus*) and with difficulty into rats and mice. Subinoculation

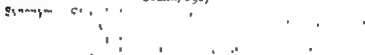
to n

It p

for

relapsing fever. Probably it is spread by the agency of lice.

**Spiroschaudinna morsusmuris** Tutsui, Takaku, Taniguchi, and Osumi, 1917



but no undulating membrane and has generally three or four curves, but may have two to nineteen. The smaller forms occur in the blood and the larger in the tissues.

Mice and white rats become affected but guinea-pigs and monkeys fail to do so.

**Spiroschaudinna icterohæmorrhagica** Inada, Ido, Hoki, Kaneko, and Ito, 1915

**Synonyms.**—*Spirochate icterohæmorrhagica* Inada, etc., 1915; *S. icterogenes* Uhlenhuth and Fromme; *S. nodosa* Huebener and Reiter.

**Nomenclature.**—Noguchi has created a new genus (see p. 439) for this parasite, calling it *Leptospira icterohæmorrhagica*, and this will probably be generally accepted.

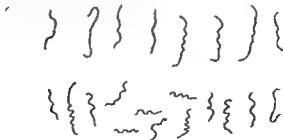


FIG. 133.—*Spiroschaudinna icterohæmorrhagica* Inada, etc.

This genus . . . . .

by "

by

by ... (1882)

onchitis in  
Gantham

*S. bronchialis* is an organism with marked polymorphism varying in length from 5.25 microns and in breadth from 0.2-0.3 micron. The variations indicate different stages of growth and division. As a rule the ends though varying considerably are acuminate.

T ... effective agent and spread the in ...  
... from *S. dentium* and *S. buccalis*  
the mouth spirochætes of which the former measures 4.10 and the latter 9.22 microns in length.

Chalmers and O'Farrell's experiments tend to show that monkeys can be infected.

#### *Spiroschaudinna minuta* Castellani 1916

Found in cases of rhinopharyngitis (p. 1831). With Romanosky it stains a pinkish red and has very few spirals. Length 3.10-12 microns.



FIG. 136.—*Spiroschaudinna minuta* CASTELLANI

#### D. ALIMENTARY CANAL SPIROCHÆTES

#### *Spiroschaudinna subtilis* Castellani 1907

Found in scrapings from oral mucosa and in intestinal contents

It is accord n<sup>o</sup> to Prowazek  
 transmit  
 it is c  
 Variants

*Spiroschaudinnia aboriginalis* Cleland 1909

Th. a.

6.5 μ opbyte

✓ 3 4 4 0 3 inguinale but merely a

*Spiroschaudinnia phagedenis* Noguchi 1912

N

*S. acuminata* Castellani 1905 and *S. obtusa* Castellani 1905

Found in the open sores of yaws

*S. pseudopalida* Mulzer 1905

Found in ulcerat<sup>o</sup> carcinomata

Unfixed forms of cutaneous spirochaetes have been seen by von Prowazek in cases of psoriasis

C RESPIRATORY SPIROCHETES

*Spiroschaudinnia bronchialis* Castellani 1907

The presence of this spirochete and th



FIG 134 AND 135 — *Spiroschaudinnia bronchialis* CASTELLANI



## THE DIFFERENTIATION OF HUMAN SPIROCHÆTES

The differentiation of spirochætes is exceedingly difficult morphological characters seldom help and measurements with variations are useless for this purpose

As many of the so-called species are probably only variants produced by environment it seems correct to classify them according to site in the human body according to their action on man and animals and according to immunity experiments

The following is an attempt on these lines —

- A Found in the blood —**
- I In cases of relapsing fever —
    - (a) Clinical symptoms in man mild but in animals severe —  
—*S novyi*
    - (b) Clinical symptoms in man severe —
      - 1 In animals severe—*S duttoni*
      - 2 In animals mild —
        - (1) Berbera immune serum protective—*S berbera*
        - (2) Berbera immune serum ineffective and currentis immune serum protective close allied forms —
          - (a) Found in Europe—*S recurrentis*
          - (b) Found in India—*S carteri*
    - II In cases of infectious jaundice—*S icterohæmorrhagica*
    - III In cases of rat bite disease—*S morsusmuris*

**B Found in the skin —**

    - I In ulcus tropicum —  
Not cultivated with undulating membrane and short flagellum—*S vincenti*
    - II In granuloma inguinale —  
Not cultivated without undulating membrane or flagellum —  
—*S aboriginis*
    - III In cutaneous inflammation —
      - (a) Cultivated strictly anaerobic causes transient inflammation in animals—*S phagedenis*
      - (b) Not cultivated found in open yaws ulcers —
        - 1 Acuminate—*S acuminata*
        - 2 Obtuse—*S obtusa*
      - (c) Not cultivated found in ulcerating carcinomata—  
*S pseudopallida*

**C Found in the respiratory passages —**

    - I In bronchial spirochaetosis—*S bronchialis*
    - II In rhinopharyngitis—*S minuta*

**D Found in the alimentary canal and skin lesions —**

    - I In the mouth —
      - (a) Produces pseudo-membranes in cases of angina and ulcus tropicum—*S vincenti*
      - (b) Non pathogenic —

**Spiroschaudinnia curygyrata** Werner 1909 *emendavit* Fantham 1910

**Synonyms**—*Spirillum hachatzæ* Kowalski (perhaps) *S curygyrata* Werner 1909

The spirochæte was first noted under the name spirillum in cholera motions in 1884-86 and 1893-94. In 1903 Le Dantec saw them in cases of so-called spirochætal dysentery. In 1909 Werner gave them the two names mentioned above which Fantham in his 1910 researches considers to be the same.

The spirochætes were found by Fantham in the fæces of soldiers

bodies

**NOTE**—It is essential not to confuse true spirochætes found in the fæces with vegetal organisms with pseudo spirochætal shapes as has occurred—e.g. *Spirobacillus* (*Bacillus*) *eylanicus* Castellani 1910 has often been mistaken for a true spirochæte (see p. 1838)

## C. URINARY SPIROCHÆTES

**Spiroschaudinnia urethræ** Macfie 1917

These spirochætes have been seen by Macfie in urine in natives on the Gold Coast where it caused an acute urethritis.

The parasite was found free in large quantities in the pus and also in the pus cells. It measured from 5-20 microns in length and

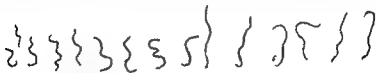


FIG. 137.—*Spiroschaudinnia urethræ mitis* CASTELLANI

showed a membrane or crest. The cytoplasm which was homogeneous contained chromatin granules or rodlets at intervals. Some pus cells contained coccoid granules.

Spirochætes have been found in the normal urethra by various authors—e.g. Mendelsson.

*S. mitis* was found by Castellani in the centrifuged urine of certain cases of camp jaundice of mild type. It is thicker than *S. icterohæmorrhagica* and has larger and better defined waves. Of doubtful pathogenicity.

Recent researches by Balfour tend to show that the Sudan strain is a separate species *S grandiosa* Balfour 1910 Aragão has attempted to obtain a serum and a vaccine with a certain degree of success

**Spiroschaudinna neveuili** Brumpt 1909

Brumpt describes this spirochæte as morphologically identical with *S marchouxii* but cross immunization shows that the two species are different It is the cause of foal spirochaetosis in Senegal and is spread by *Argas persicus*

**Spiroschaudinna theileri** Laveran 1904

This spirochæte discovered by Theiler in 1902 is found in cattle in Africa about Pretoria in the Cameroons and in East Africa The symptoms are not clear as babesia has also been seen in the same animals It is spread by *Margaropus decoloratus* the blue tick

**Spiroschaudinna ovina** R Blanchard 1906

This spirochæte may be the same as *S theileri* It was found by Marfoglio and Carpano in sheep in Erythræa on the Red Sea and by Theiler in the Transvaal

**Spiroschaudinna equi** Novy and Knapp 1906

Found by Theiler in the Transvaal and by Martin in French Guinea and may be the same as *S theileri*

Other Spirochætes — *S vesperisomus* Novy and Knapp found by Nicolle

ilev

.. .. ind

Prowazek in balantids

*S litorea* Prowazek 1907 found by Prowazek in the otter

**Treponemidæ** Schaudinn 1905

**Definition** — Spirochætaea with a minute thread like body

**Classification** — Only one genus

**Treponema** Schaudinn 1905

**Synonym** — *Spirochæta* Ehrenberg *pro parte* *Spironema* Vuillemin 1905 von Klebs 1892

Treponemidæ with the characters of the family

**Type Species** — *Treponema pallidum* Schaudinn 1905

1. Short forms—*S. dentium*.

2. Long forms—*S. buccalis*

II. In vomit —

Rather doubtful forms in vomit of Belyando spew in Queensland—*Unnamed*

III In fæces —

In health and disease—*S. eurygyrata*.

E. Found in the urethra —

I In free and in coccoid form in pus cells from urethritis. With crest or membrane—*S. urethrae*

II In urine from cases of mild camp jaundice. With well-marked waves—*S. nutis*

## ANIMAL SPIROCHÆTES

*Spiroschaudinnia macaci* Castellani and Chalmers 1910

**Synonym.**—*Spirochæta macaci* Castellani and Chalmers, 1910.

This spirochæte was found by us in monkeys in Ceylon in 1906. In length it measures about  $13 \mu$ , and closely resembles *S. carteri* Manson, 1907. It can be easily inoculated from monkey to monkey. Spirochætes which may be of a different species have also been found by Leishman, Balfour, and by Plimmer in *Cercopithecus sebæ* from Sierra Leone.

*Spiroschaudinnia anserina* Saccharoff  
1891

Found in enormous numbers in the blood of geese in the Caucasus and Tunis. It causes fever, diarrhoea, tenderness of the feet and death in about a week, the mortality being 80 per cent. It can be inoculated into other geese.

*Spiroschaudinnia marchouxii* Nuttall  
1904

**Synonym.**—*Spirochæta gallinarum* R. Blanchard 1905.

This spirochæte which has been discovered by Marchoux and Salimbeni and studied by Balfour, is about 10 to  $20 \mu$  in length, causes disease in geese.

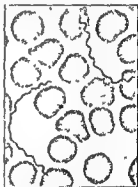


FIG 138—SPIROCHÆTES IN THE BLOOD OF *Cercopithecus sebæus* FROM SIERRA LEONE ( $\times 1000$ )

(From a microphotograph by H. G. Plimmer.)

of the whole body. The periplast is continued as long delicate processes at each end which are considered by some to be flagella.

Krzyształowicz and Siedlecki say that not far from the middle

and conjugation which they think leads to the formation of a cyst or spore, which may be carried via the blood stream to different

ta  
the secondary  
ptions though  
the placental

ens) which may be met with on trichotex surfaces by difficulty in staining number character and permanence of the spirals the terminal prolongations absence of an undulating membrane minute size and delicacy

**Inoculation**—Syphilis can be inoculated into chimpanzees (as shown by Metchnikoff and Roux) and other monkeys and *T. pallidum* can be found in the lesions so caused the incubation

and HULLMAN also cultivated it *in vitro* in maintaining a pure

a cutaneous reaction can be obtained in syphilitic patients

**Life-History**—Unknown

*Treponema pallidum* Schaudinn 1905

Synonyms—*Spirochaeta pallida* Schaudinn 1905 *Spironema pallidum* Vuillemin 1905 *Microspironema pallidum* Stiles and Dole 1905

in  
in  
a  
Dohle in 1901 described parasites and saw cell inclusions in the disease and in 1905 Siegel described in organism *Cytoryctes luis* in which there are flagellate bodies very closely resembling spirochaetes. In 1905 Schaudinn after investigating and reporting



FIG 139—*Treponema pallidum* SCHAUDINN, 1905  
(From a microphotograph by J J Bell)

unfavourably on Siegel's work for the *Treponema*

$\mu$  in length average  $7 \mu$   
from six to twelve and more in number the average being eight  
ten and are to a certain extent preformed— $\rightarrow e$  not due to the  
parasite movements. It moves by rotation on a long axis by  
ding movements forwards and backwards and also by flexion



p 409]

Pathogenicity — *T pallidum* is the cause of syphilis

**Treponema pertenue** Castellani 1905

Synonyms — *Spirochaeta pertenue* Castellani June 1905 *Spirochaeta pallidula* Castellani November 1905

History — It was discovered by Castellani in 1905 in the scrapings from yaws papules

Morphology — *Treponema pertenue* is an extremely delicate spiral shaped organism varying in length from a few microns to 18 and 20  $\mu$  and even more. It is very slender. Some individuals are however thicker than others. It does not stain easily but

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the extremities may present a large pear shaped expansion or a loop like formation. The number of coils varies from six to twenty or more but they are as a rule numerous uniform and of small dimensions. Occasionally a portion of the *Treponema* shows numerous close uniform coils while the rest of its body shows no coils at all. Sometimes two *Treponema* may be attached end to end or apparently twisted together. Castellani has not been

with flagella

**Intracellular Stage** — Castellani in 1905 described some peculiar bodies free and intracellular in leucocytes which possessed an oval or roundish shape and contained chromatin dots. At the time he was inclined to consider them to be stages in the development of the parasite but later he held that they were cell inclusions of non-



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A most complete account of the recent literature on this subject is contained in the *Kala Azar Bulletin* or the *Tropical Diseases Bulletin* and in Laveran (1917) *Leishmanioses* Paris.

- ARCHIBALD R G (May 1913) An Interesting Case of Kala Azar *Journal of the Royal Army Medical Corps* (November 1914) A Preliminary Report on some Further Investigation of Kala Azar in the Sudan *Journal of the Royal Army Medical Corps*

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*Sleeping Sickness Bulletin* (1909 1914) *1101 CALIFORNIA CASES BULLETIN* (1912 1919)

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362

## Spiroschaudinna recurrentis

- MANTEUFEL (1907) *Arb a d Kaiserl Gesundh Bd xxvii Heft 2 326*  
 OBERMEYER (1873) *Centralb f die Med Wiss*  
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- BREINL (1907) *Annals of Tropical Medicine and Parasitology* No 3  
 BREINL AND KINGHORN (1906) *Memoir XXI Laverpool School of Tropical Medicine*

spleen lymphatic glands and bone-marrow. In the blood it has not yet been demonstrated microscopically though there is no doubt that the blood of the general circulation is infectious inasmuch as monkeys inoculated with it develop typical yaws lesions, in which the *Treponema* is abundantly present. The *Treponema* is absent in the cerebro spinal fluid and generally in the tertiary lesions.

*Bacteriological Flora found in Open Sores of Framboesia*—While

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f  
h

varying in size and number and with blunt extremities—*S. obtusa* Castellani. A third form is likewise thin and delicate but tapers at both ends—*S. acuminata* Castellani. *T. pertenuis* is also present in many cases.

In 1848 Paullet taken from framboesia the inoculation when at the seat

The second night of them

followed by all the usual types of secondary eruption that

that monkeys are susceptible to framboesia. According to their experiments the inoculation period varies from a minimum of sixteen days to a maximum of ninety two. The appearance of

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## CHAPTER XX

### DIPLOZOA AND OCTOMITIDÆ

Preliminary—Diplozoa—Octomitidæ—Octomitus—Giard 1—References

#### PRELIMINARY

We now return to the classification of the Protomonadina given on p. 332 where the order is divided into two suborders Monozoa and Diplozoa. The Monozoa we have just considered and in the present chapter the Diplozoa are described.

#### SUBORDER 2 DIPLOZOA Hartmann and Chagas 1911

**Definition**—Protomonadina with more or less tendency to bilateral symmetry in *undividing forms* as shown by the arrangement of the flagella by the duplication of the axostyle with sometimes the nucleus and more rarely of the cytostome. An undulating membrane is absent.

**Classification**—This suborder may be divided into two families of which one is of importance in tropical medicine.

A Cytostome single or absent flagella eight in number

Family 1 *Octomitidæ* Minchin 1912

B Cytostome double flagella variable in number—Family 2

*Distomatidæ* Senn 1900

Only the first of these families concerns us.

#### FAMILY 1 OCTOMITIDÆ Minchin 1912

**Definition**—Diplozoa with eight flagella and with or without a cytostome.

**Type Genus**—*Octomitus* Prowazek 1904

**Classification**—The family is divisible into several genera as follows—

A Anteriorly three pairs and posteriorly one pair of flagella  
nucleus single bilaterally lobed or doubled sucker  
absent

I Parasitic—*Octomitus*

II Free living—*Hexamita*

Octomitus and Giardia concern us. Hexamita does not

chool of Tropical Medicine  
dicine  
stitut Pasteur xvi  
vol ii p 1453

**Spiroschaudinnia carteri**

- CARTER (1882) *Spirillum* Fever London  
MACKIE (1907) *Lancet* ii September 21 and December 14

**Spiroschaudinnia bronchialis**

- icale (1917)  
\*medicine ind  
xiv 391  
VILLÉ (1915) *Bull Path Exot and Lancet*  
VIOLE (1918) Bronchite sanglante (Spirochétose broncho pulmonaire de  
Castellani) *Presse Médicale* No 39

**Spiroschaudinnia in Yellow Fever**

- NOGUCHI (1919) *Journal American Medical Association* January 18

**Spiroschaudinnia heterohæmorrhagis**

- INADA IDO HOKI HANEAO ITO (1916) *Journal of Experimental Medicine*  
xxviii p 377  
MARTIN AND PETIT (1917) *Comptes Rendus Soc Biol*  
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**Treponema pallidum**

- KRYSZTAŁOWICZ AND SIEDLECKI (1906) *Bull Inst Past* vol iv p 204  
{Abstract}  
NOGUCHI (1910 1918) : Several important papers in the *Journal of Experi*  
ed

**Treponema perenne**

- ASHBURN AND CRAIG (1907) *Philippine Journal of Science* vol ii p 441  
CASTELLANI (June 1905) *Journal Ceylon Branch British Medical Association*

veral papers

†

in the *Journal of*

*Medicine*

In front of the nucleus and closely approximated to the anterior end of the body is a well defined blepharoplast which though typically single may have a secondary smaller blepharoplast as

th

br

arise from each

These flagella vary considerably in length being generally much longer than the body but it is exceedingly difficult to be certain as drawn only such portions as though in other specimens the in some instances quite three

ising from the blepharoplast or blepharoplasts there are two chromatic lines which diverging and running on either side of the nucleus may or may not converge but in either case end near the posterior margin of the body in very minute chromatic particles which are generally very difficult to see From each of these chromatic particles there arises a posteriorly directed flagellum

The chromatic rods are obviously axostyles and the little particles in which they end may be termed axoplasts

It is not often that the axostyles are seen together as the parasite usually lies so that only one is visible while the other is almost invisible but at times they are seen as described above or at other times when the parasite has shrunk into a rounded mass they may be observed crossing one another

No cytostome has been observed while the periplast is thin and without markings

The cytoplasm is vacuolated with food vacuoles

**Life-History**—Nothing is known of the life history

**Pathogenicity**—Believed to cause diarrhoea

### Genus *Giardia* Künstler 1882

**Synonyms**—*Lamblia* R Blanchard 1888 *Dimorphus* Grassi 1879 *nec* Haller 1878 *Megastoma* Grassi 1881 *nec* de Blainville

**Definition**—Octomitidæ with one antero mesial pair two pairs of mesial and one pair of posterior flagella nucleus usually double sucker present

**Type Species**—*Giardia intestinalis* (Lambl 1859)

### *Giardia intestinalis* (Lambl 1859)

**Synonyms**—*Lamblia intestinalis* Lambl 1859 *Cercomonas intestinalis* Lambl 1854 *Hexamitus duodenalis* Davaine 1875 *Dimorphus muris* Grassi 1879 *Megastoma entericum* Grassi 1881 *Megastoma intestinale* Blanchard 1886 *Lamblia intestinalis* Blanchard 1888

It is parasite lives in the intestine of different species of the

## GENUS I OCTOMITUS PROWAZEK 1904

**Definition**—Octonutidæ parasitic with anteriorly three pairs and posteriorly one pair of flagella nucleus single bilaterally lobed or doubled sucker absent

**Type Species**—*O. intestinalis* Prowazek 1904 found in rats

**Classification**—The known species of *Octomitus* may be recognized as follows—

A

∴  
∴

(a) Measurements 8.12 × 5.7 microns—*Intestinalis*

(b) Measurements 4.6 × 2 microns—*Muris*

B Nucleus not situate close to the anterior end—

Nucleus single and rounded size 6 × 4 microns—*Hominitis*



FIG 143—*Octomitus hominis* CHALMERS AND PEKKOLA 1916  
(× 2000)



FIG 144—*Octomitus hominis* CHALMERS AND PEKKOLA 1916  
(× 2000)

*Octomitus hominis* Chalmers and Pekola 1916

**Definition**—*Octomitus* with single rounded nucleus not situated close to anterior end

**Morphology**—Small fusiform or pear shaped flagellites in size 5.660 × 2.830 microns

When examined in the stained condition it showed a circularly defined nucleus (Figs 143 and 144) measuring about 1.4 microns in transverse diameter and lying near the junction of the anterior third with the posterior two thirds of the body. The nucleus is bounded by a well marked membrane which limits the homogeneous dark staining contents in which there is often

In addition in the living animal refractile granules can be seen lying in the anterior part of the animal on each side of the mesial line

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L . . .

The process is very complicated the whole system of sucking disc and flagella being reproduced dorsally and then the flagellate splits longitudinally, the fissure passing from before backwards between the sucking discs

Kofoid and Christiansen have described multiple fission in the lamblia of mice but this has not been seen in those in man

inside which the  
becomes ovoid and  
or may not have  
occur

The cysts are oval and measure 13 14 x 6 / microns the wall is smooth and transparent Later the nuclei divide giving rise to four nuclei in all which are crowded together

**Method of Infection**—The cysts escape in faecal matter and are taken into house flies *Musca* and *Fannia* etc and passing into the intestine eventually escape in the flies droppings and so can infect human food

**Pathogenicity**—It is usually believed to be the cause of the diarrhoea with which it is associated

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June 15 142 146 London

##### *Giardia intestinalis*.

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genera *Mus* and *Epimys* (*M. musculis* F. v. r.

It is  
 found in  
 encysted  
 and as it occurs in mice it is quite easy to see how infection of  
 foodstuffs experimentally by Grassi

G  
 about  
 in br... this pear shaped being surrounded  
 by a thin periplast (ectoplasm) which keeps  
 its form. The under surface (when attached)  
 is excavated with a well defined border which  
 is interrupted at the site of the cytostome.  
 This hollow is probably a kind of peristome  
 and is useful in fixing the parasite to the  
 intestinal epithelium.

There are two oval nuclei with definite  
 nuclear membranes and with large irregular  
 karyosomes in their centres. There is no  
 connection between these nuclei but between  
 them lie two darkly staining rods with ex-  
 tended ends and which posteriorly are con-  
 tinuous with the prolongations of the posterior  
 flagella into the body. From the thickened  
 posterior ends of the two rods spring the mesial pair of flagella  
 while at their anterior ends is a small granule from which arises  
 the anterior pair of flagella which running forwards and inwards  
 cross one another and pass across the peristome or sucking disc  
 or sucker to its raised margin around which they run forming a  
 band of membrane till nearly at the level of the nucleus they  
 become free on each side.  
 From the same anterior part of ...



FIG. 145 — *Giardia intestinalis* (LANDEL, 1859) (After Wenyon)

Sometimes there is a row of granules extending from ...



**Coccidium schubergi** Schaudinn, 1900

*Coccidium schubergi* begins its life-history in the intestinal cell of the centipede (*Lithobius forficatus* L.) by a sporozoite pressing its anterior end against



FIG 146—DIAGRAM OF THE LIFE HISTORY OF *Coccidium schubergi* SCHAUDINN (After Schaudinn)

1, Sporozoite entering an intestinal canal 2 3 trophozoites, 4 5 schizonts, 6 8, merozoites, 9 young gametocyte 10 11a microgametocyte, 10b-11b macrogametocyte, 12a microgametes 12b macrogamete 13-16 oocyst, 17 sporocysts, 18, sporozoites

## CHAPTER XXI

# TELOSPORIDIA

Telosporidia—Gregarinida—Coccidiidae—Hæmosporidia—Hæmogregarinidæ  
 — Toxoplasmidæ — Piroplasmidæ — Plasmodiæ — Hæmoproteidæ —  
 References

### PHYLUM III TELOSPORIDIA Schaudinn 1900

**Synonym**—*Eimeriinea* Poche 1913

**Definition**—*Parasitic plasmodia* without motile organs in which the reproductive phase of the life cycle which produces spores is distinct from and follows after the trophic phase

**Remarks**—All the Telosporidia are parasitic and usually begin their life cycle as small amœboid bodies with a single nucleus called trophozoites which absorb nutriment and grow and when fully developed show a cuticle an ectoplasm and an endoplasm. The endoplasm is granular and contains a vesicular nucleus with chromatin karyosomes. The nucleus of the fully grown trophozoite now begins to divide and the parasite is known as a schizont

which instead of developing into trophozoites become sexual gametocytes male and female. These forms, which are often resistant are the means of transmitting the given species from one host to another. They produce gametes which conjugate and form  
 I into three orders  
 II the two latter

the e may be recognize I as follows —

- A Only young trophozoites intracellular—*Gregarinida*
- B Full trophozoite stage intracellular—*Coccidion orpha*
  - I With resistant spores in the sporocysts—*Coccidiidae*
  - II Without resistant spores in sporocysts—*Hæmosporidia*

### ORDER I GREGARINIDA Lankester 1866

**Synonyms**—*Gregaridea* Lankester 1895 *Gregar* & Hæckel 1866

**Definition**—Telosporidia in which only the young trophozoites are intracellular the fully grown forms being extracellular

**Elmeria** A Schneider 1875

*Tetrasporocystidæ* with the formation of an oocyst after fecundation spore blasts in the form of a pyramid spores globular or oval provided with a micropyle

*Elmeria stiedæi* Lin Lemann 1865

**Synonyms** — *Psorospermus cuniculi* Rivolta 1878 *Coccidium oviforme* Leuckart 1879 *C perforans* Leuckart *Pfeifferia princeps* Labbé 1896

This is the common species found in the liver of rabbits The spores

**FAMILY POLYSPORO-CYSTIDÆ** Leger

Comprises a large number of genera *Adelia* A Schneider 1875 (dizooic) *Alossia* (tetrazooic) A Schneider 1875 *Mischusia* Labbé 1896 *Alossiella* Smith and Johnstone 1902 *Barronsia* (monozooic) *Benedenia* (trizooic)

**Coccidiosis in Man**

The utmost confusion has existed as to this infection of man but now thanks to the labours of Dobell whose writings we have followed the subject is more defined

In 1841 Johannes Muller introduced the name psorosperms for the spores of the mycosporidia and as the coccidia were believed to resemble these bodies they were called oviform psorosperms

arious  
time

**Diseases now known not to be Coccidiosis** — These are (1) blastomycosis (granuloma coccidoides) (2) rhinosporidiosis

Pitres (1884)

\* Cases correctly recognized as Coccidiosis — Dobell considers that over seventy cases mostly from the Near East have recently been recognized as coccidiosis The earlier cases are —

*Hepatic* — Grubler in Paris (1858) Dressler in Prague (recorded by Leuckart in 1863) Sattler in Vienna (recorded by Leuckart in 1879) Perls in Glessen (recorded by Leuckart in 1879) Perls and von Sommering (?) (recorded by Leuckart in 1879) and Silcock in London (1890)

Some merozoites now become differentiated into micro and macro gametocytes. The former consist of finely granular cytoplasm with little reserve material while the latter are bean shaped and have much food

gamete

- FAMILY 2 DISPOROCYSTIDÆ —Oocyst has two spores  
 FAMILY 3 TETRASPOROCYSTIDÆ —Oocyst has four spores  
 FAMILY 4 POLYSPOROCYSTIDÆ —Oocyst has many spores

#### FAMILY ASPOROCYSTIDÆ Leger 1900

No genus included in this family is *Eimeria* but there is only one species *Eimeria* in a Schneider 1891 the Malpighian tubules of *Glomeris*

#### FAMILY DISPOROCYSTIDÆ Leger 1900

The genera of this family are *Cylicospora* A S Schneider 1881 (sporozoite)  
*Diplospora* Labbe 1893 (spores tetrazoic) *Isospora* A S Schneider 1881 (spores polyzoic)

*Isospora* A Schneider 1881

Definition —Disporocystidæ with polyzoic spores

*Isospora bigemina* Stiles 1891

man *infra*)

#### FAMILY TETRASPOROCYSTIDÆ Leger 1900

Type Genus —*Eimeria* A S Schneider 1874

Synonym —*Coccidium* Leuckart 1879 (the dizoic spores are spherical or oval) Other Genus *Crypsall* spores Labbe 1893 (the dizoic spores have the form of a double pyramid)

microns External surface of sporocyst rough No oocystic residual body Each spore contains two typical sporozoites and one or two sporocystic residua Habitat man

**History**—This parasite was found by Woodcock and Wenyon in 1915 in the feces of a British soldier from Gallipoli It was again found by Roche in 1917 in three cases at Salonika so that the total infections up to date (1918) are four

**Distribution**—Shores of the Eastern Mediterranean

**Pathogenicity**—Unknown and no attempts so far made to infect animals

#### *Eimeria oxyspora* Dobell 1918

**Definition**—*Eimeria* with spherical oocyst 36 microns in diameter with faintly yellow transparent wall composed of at least two distinct layers containing four dizoic spores and a small oocystic residue Spores long sharply pointed at both ends  $30.32 \times 7.5$

posterior ends which contain the nucleus

**History**—The parasite was found by Dobell in a young man who had been in South Africa Ceylon and India

**Distribution**—Unknown

**Pathogenicity** Infection small but pathogenicity not certainly known because the patient was infected with *L. histolytica* and *Ancylostoma* Believed not to be pathogenic

#### The Hepatic Coccidium of Man

**Synonyms**—*Cellules ovoides* (?) *œufs d'helminthes* Gubler 1858  
*Corps ovoides* Davaine 1860 *Psorospermien* Leuckart 1863  
*Psorospermi* Rivolta 1873 *Coccidium ovoides* Leuckart 1879  
*Coccidium leberpsorospermien* Butschli 1882 *Coccidium cuniculi*  
 (Rivolta) Blanchard 1896 *Eimeria stiedæ* (Lindemann) Luhe 1906  
*Eimeria* (?) sp Dobell 1918

**Definition**—Not at present capable of definition

**History**—It was first recorded by Gubler in 1858 in a quarryman aged forty five in Paris This man is said to have died from peritonitis He suffered from digestive troubles anæmia and had an enlarged liver Post mortem the liver contained many tumours

were completely filled with granular contents One end was flat blunter than the other which showed a slight constriction and had a small depressed surface as though an operculum or micropyle were present

The second case was found by Dressler of Prague and consisted of three small nodules in the margin of a human liver These nodules contained a whitish pulp which surrounded oval bodies 18-20 microns

*Intestinal*—Kjellberg (recorded by Vitchow in 1860) and two cases by Eimer (1870)

Lc

is (1917) Martin  
and Boulenger

the above cases

*Isospora hominis* Rivolta 1878 *emendat* Dobell 1913

Synonyms—*Psorospermien* Vitchow 1860 Leuckart 1863 Eimer 1870  
*Cytospermis*  
*Cocidium*  
*ferans* var  
Labbé 18  
*baginina* Stiles 1891 *pro parte* I uhe 1906

Definition—*Isospora* with oocysts elongate ovoid in form narrow end drawn out into a neck 25.3 x 12.5 x 16 microns with clear colourless and porcellaneous wall with two or more layers and an inconspicuous micropyle at narrow end Development of spores completion sporocysts granular

not 1860 in  
Eimer and

was named in 1878 by Rivolta Its oocysts were probably first found in human faeces by Railliet and Lucet in 1890 but the first clearly recognizable account is that given by Wenyon in 1915 since when some fifty cases of infection have been recorded making in all with the cases seen by Castellani and Richards in the Balkans about seventy infections

Dobell +

*Eimeria wenyoni* Dobell 1918

Synonyms—*Eimeria* (*Coccidium*) Wenyon 1915 *Cocidium*

A. *Without hæmozoin* :—

- I. Live in red and white blood cells in the peripheral blood—*Hæmogregarinidæ*.
- II. Live in white cells in the organs—*Toxoplasmidæ*.
- III. Live in red cells in the peripheral blood—*Piroplasmidæ*.

B. *With hæmozoin* :—

- I. Ookinete encysts and forms an oocyst—*Plasmodidæ*.
- II. Ookinete is not known to encyst—*Hæmoprotozoidæ*.

## FAMILY HÆMOGREGARINIDÆ Neveu-Lemaire, 1901.



obtained

The evolution of the knowledge concerning these parasites may be briefly stated

itself

The term *Drepanidium* having been previously employed for one of the Heterokaryota, it was necessary to alter it to *Lankasterella*, and the term *Danilewskya* was also altered by Danilewsky in 1897 to 'Hæmogregarina' (*sensu stricto*), but since Sambon and others have described so many new

in length and his drawings show four oocysts. The third case was discovered by Sattler of Vienna in a pathological preparation. It showed a dilated bile-duct with greatly proliferated epithelium and coccidia.

The fourth case is by Perls; it was from a preparation made by von Sommering and is said by Leuckart to have contained coccidia.

The fifth and last case is that described by Silcock in 1850 at St. Mary's Hospital, London.

surrounded by an inflammatory zone and the large intestine had deeply congested patches of mucosa.

In the caseous nodules were agglomerations of small oval egg-like bodies with granular contents and a well marked capsule and were considered to be identical with Leuckart's coccidia. They were kept in water and sporosperms freely developed. He considered them to be *Coccidium oviforme* and to be present in the spleen but does not state anything definite as to the intestine.

In all therefore up to 1918 five cases have been recorded.

Dobell does not consider this parasite to be *Eimeria stiedei* judging from Dressler's drawings.

Gubler's case was considered to be a hydatid cyst. Dobell's conclusions are that there is a coccidial parasite which very rarely occurs in the human liver and resembles *E. stiedei* but is considerably smaller and is probably a distinct species though perhaps belonging to the same genus.

**Distribution**—Europe.

**Pathogenicity**—It causes cyst like swellings of the liver with enlargement of that organ.

### ORDER III HÆMOSPORIDIA Doflein 1901

**Synonym**—*Hæmocytozoa* Mesnil 1915

**Definition**—Telosporidia Coccidiomorpha without resistant spores in the sporocysts and with the trophozoite stage intracellular. With alternations of generation schizogony in a vertebrate and sporogony in a blood sucking arthropod or leech.

**Remarks**—Mesnil considers that the family Hæmosporidiaceæ is related to Leger's Adeleidea division of the Coccidiales and the Plasmodiæ to the Eimeriada division and that they should find their places in that group. He considers that the genus *Leucocytozoon* should come into the Hæmosporidia and that the *Leucocytozoon*

in 1915

T

17



comes  
"

With regard to the Hæmogregarinida of reptiles a very complete work is that of Sambon in 1908 on the parasites in snakes where a full history will be found. Those which occur in tortoises and crocodiles have been studied by Danilewsky, Castellani and Willey, Miss Robertson, Dobell and

the cycle of schizogony while others enter mononuclear leucocytes in which they become encysted and develop into gametocytes.

traced. A danger is to mistake *Crithidia pulvis* of the flea for a developmental stage of the *H. jaculi*.

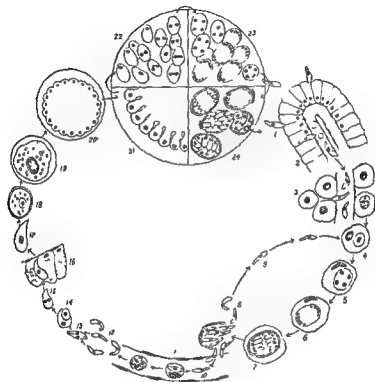


FIG 148.—LIFE CYCLE OF *Hamogregarina in isis* BALFOUR  
(After Miller)

1, 2, Trophozoites  
entering  
9 merozoites  
blood cells  
17 the  
the stomach  
sporoblasts

In 1906 Adie discovered a hamogregarine in *Epimys rufus* Balfour one in *I. norvegicus*, Christophers another in *Ictis domestica*, Patton another in *Funambulus pennantii*.

In 1907 Christophers traced the sporogony of *H. canis* in *Rhipicephalus sanguineus*. In 1908 Miller contributes a most valuable paper on *H. muris* Balfour, 1905, under the term *Hepatozoon perniciosum* Miller, 1908, in which he

cell as an oval fusiform or club shaped mass of cytoplasm with a



FIG 15.—*Hæmogregarina mirabilis* CASTELLANI AND WILLEY SHOWING THE ESCAPE OF THE GAMETOCYTE FROM ITS CAPSULE  
(After Castellani and Willey)

This differentiation is considered to be a prelude to the formation of m...

the micro gametocyte



FIG 154.—*Hæmogregarina seligma ins*  
SAMBON FREE SPORONT  
(After Sambon)



FIG 155.—*Hæmogregarina seligma*  
SAMBON  
According to Sambon this figure probably represents conjugation  
(After Sambon)

Infection of the rat takes place by ingestion of the mite when the sporozoites are liberated by the juices of the duodenum and become actively motile striated vermicles which penetrate the intestinal villi enter the blood stream and are carried to the liver into the cells of which they penetrate and start the cycle of schizogony

As the mites leave the rats during the day time and only feed on them during the night it is easy to understand the manner in which the disease spreads from the sick to the healthy

**Variations in the Life-Cycle —**

The life history of *H. muris* is peculiarly interesting because it is fully known but it is not quite typical for all hæmogregarines for in it the gametocytes alone are found in the peripheral blood and they are enclosed in leucocytes

Other species however show marked differences from *H. muris* for the majority are found in red



FIG 149 — *Hamogregarina vittata* ROBERTSON SHOWING SCHIZOGONY

(After Miss Robertson)

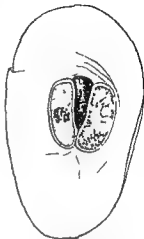


FIG 150 *Hamogregarina rarefaciens* SAMBON SHOWING MALE AND FEMALE SCHIZONTS

(After Sambon)

not white corpuscles while in some the whole process of schizogony is completed in the blood stream therefore a few more general remarks are necessary to supplement the life history given above



FIG 151 — *Hamogregarina rarefaciens* SAMBON YOUNG GAMETOCYTE

(After Sambon)



FIG 152 — *Hamogregarina stignani* SAMBON SHOWING CAPSULE AND CLEAVAGE LINES

(After Sambon)

The peripheral blood of the vertebrate can contain trophozoites schizonts and gametocytes



on an ordinary slide. The further history of the cycle of sporogony is only known in *H. muris* and *H. canis*; the former has already been described and the latter will be mentioned later.

Recently Henry has shown that Balfour's infective granule is a phase in

in the spleen of a Chinese

**Classification.**—As already mentioned the species of the genus *Hæmogregarina* Danilewsky 1885 will be arranged according to their hosts. Some authors recognize *Hepatozoon* Miller 1908 with *H. muris* Balfour 1905 as a type and distinguish it by living in leucocytes and sometimes undergoing schizogony in the cells of the internal organs.

## HÆMOGREGARINES OF THE MAMMALIA

### *Hæmogregarina hominis* Krempf 1917

Parasite of man

" "

### *Hæmogregarina muris* Balfour 1905

**Synonyms.**—*Leucocytozoon muris* Balfour 1905; *Hepatozoon perniciosum* Miller 1908

of *Epimys norvegicus* in  
C. Its schizogony and  
described (p. 479). It

### *Hæmogregarina canis* James 1905

Synonym — ?



zoite grows into the encapsuled form just described, all stages between the two having been seen by Christophers and in this way the cycle of schizogony is completed

Sporogony — The tick *E. canis* on the dogs in Madras the blood which it only taken in the last twenty and when examined at made out —

The encapsuled forms already described as existing in the blood pass into the stomach and the parasite escapes from the corpuscle but is still inside its own envelope.

By elongation and passage of the protoplasm behind the nucleus the oval parasite becomes a vermiform. These are as macrogony young epithelial cells divide by fission the secondary forms cytoplasm of the cells as a rule do not divide.

near the periphery

#### *Hæmogregarina bovis* Marioglio and Carpano 1906

In *Bos taurus* in Abyssinia. The parasites are 7 to 10  $\mu$  in length and 1.5 to 2  $\mu$  in breadth and possess rounded ends.

#### *Hæmogregarina gerhali* Christophers 1903

Found in the Indian field mouse. The parasites are little as with a louse // its cyst on the

*Hæmogregarina* " " "

Synonym  
This form a  
genous erythrocyte

The trophozoite is found in a liver cell as an oblong parasite lying in a cavity. This body can divide into three young forms which presumably can grow



into schizonts in liver cells. The schizont divides into a large number of merozoites leaving no residual mass of undivided cytoplasm. The merozoites probably infect the red blood cells and after a time can escape into the liquor sanguinis as free trophozoites and invade the liver cells.

What happens to the free vermicle is not known.

#### *Hæmogregarina funambuli* Patton 1906

**Synonym.**—*Leucocytozoon funambuli* Patton 1906

This parasite was found by Patton in the large mononuclear leucocytes of

the coelome but no further development took place.

#### *Hæmogregarina rattii* Adie 1906

This is a hæmogregarine found in the leucocytes of *Epmys rattus*.

#### *Hæmogregarina felis* Christophers 1906

Like *H. canis* only found in cats. It was discovered by Patton. Schizogony and sporogony unknown.

### HÆMOGREGARINES OF REPTILIA

#### Hæmogregarines of the Crocodilia

*H. hankini* Sumond 1901 in *Gattalis gangeticus* Gmel. *H. crocodilorum* Borner 1901 in *Osteolemus tetraspis* Cope and in *C. cataphractes* Cuv.

#### Hæmogregarines of the Chelonia.

A very large number of hæmogregarines are known in Chelonia but the best studied life history is that of *H. stepanovi*.

#### *Hæmogregarina stepanovi* Danilewsky 1889

*H. stepanovi* is a parasite in the red blood-corpuscles of tortoises—*Emys orbicularis* L. and *Cistido*.

It appears in two forms—one kidney shaped and the other long and thin and bent upon itself.

The young trophozoite is club shaped and grows into the broad kidney

of immature embryo leeches.

#### *Hæmogregarina meonæ* Castellani and Willey 1904

This parasite is common in the tortoises (*Nicoria trjuga* Schweigg) which are found in the ditches and marshy lands round Colombo and also in Colombo Lake. The young trophozoite grows into the schizont which divides into merozoites.

zoite grows into the encapsuled form just described all stages between the two having been seen by Christophers and in this way the cycle of schizogony is completed

made out —

The encapsulated forms already described as existing in the blood pass

#### *Hæmogregarina bovis* Marfoglio and Carpano 1906

In *Bos taurus* in Abyssinia. The parasites are 7 to 10  $\mu$  in length and 3 to 2  $\mu$  in breadth and possess rounded ends

#### *Hæmogregarina gotbilli* Christophers 1905

Found in the Indian field rat *Cerbillus indicus* in which it produces only a little anaemia. It lies in cysts in enlarged pale blood corpuscles as a vermicle

no distinct nucleus

There is however some doubt as to whether these cysts are really developed from the hæmogregarine

#### *Hæmogregarina jassali* Balfour 1905

Synonym.—*H. balfourii* Laveran 1905

This parasite has been found in the jerboa (*Jaculus gordonii*) at Khar toum and in *J. orcutti* in Tunis. It appears as a pale hyaline homogeneous body with the narrower end bent on itself lying in a decolourized erythrocyte

The trophozoite is found in a liver cell as an oblong parasite lying in a cavity. This body can divide into three young forms which presumably can grow

1912, and *T. francae* de Melo 1915, with two unnamed species by Plimmer, 1916, and five by Carini and Maciel in 1916. In snakes one species unnamed was found by Plimmer in 1916.

**Toxoplasma gondii** Nicolle and Manceaux 1908

Endolencocytic crescentic parasites 6 to 7  $\mu$  by 3 to 4  $\mu$  found in the spleen and other organs of *Ctenodactylis gondii*.

**Toxoplasma cuniculi** Splendore 1909

Found by Splendore in the spleen and other organs of the rabbit, in which it produces lesions resembling kala-azar. Shape oval or reniform length 5 to 8  $\mu$  breadth 2.5 to 4  $\mu$ .

**Toxoplasma pyrogenes** Castellani 1913

Found in the peripheral blood of a case of splenomegaly in a child on the Black Sea coast and also in the blood of a dog from the same neighbourhood.

**Morphology.**—Roundish oval or crescentic bodies 2.5 to 6.0 microns in diameter with blue staining cytoplasm and with one large roundish mass of chromatin at one pole or in the centre. In one

found in a leucocyte.

by a number of protozoologists and many of them as to the parasitic and protozoal nature of the bodies but while the majority regarded them to be *Toxoplasma* others held the view that they might represent a new genus between *Toxoplasma* and *Leishmania* and a few thought that they might be a mixture of *Toxoplasma* with *Theileria* and *Anaplasma*.

**Life-History.**—Unknown

**Cultivation.**—So far not cultivated

**Pathogenicity.**—Probably the cause of a splenomegaly in man

### Hæmogregarines of the Ophidia

The hæmogregarines of Ophidia are numerous but their life history has not been properly studied. They have mostly been described by Sambon.

#### *Hæmogregarina mirabilis* Castellani and Willey 1904 (Fig. 153)

*H. mirabilis* is a parasite in the red blood cells.

The trophozoite having no clear outline and is enclosed in a Schuffner's dot.

Other forms are — *H. pythonis* Bullet 1895 in *Python reticularis*; *H. pococki* Sambon and Seligmann 1907 in *P. molurus* L.; *H. schat* 1907 in *P. molurus* L.; *H. schat* Sambon and Seligmann 1907 in *P. molurus* L.; *H. naja* Laveran 1907 in *Naja naja*.



FIG. 157 — *Hæmogregarina villosa* ROBERTSON A BROAD FORM SHOWING PECULIAR RFD BODIES

FIG. 158 — *Hæmogregarina villosa* ROBERTSON GAMETOCYTE

(After Miss Robertson)

1902 in *Vaja irish idans* M... ..  
*ascivorus* Pa... ..  
 Sambon 19... ..  
*appocrepis* I... ..  
*ermis* L. *H*... ..  
*rarefacien*... ..  
 Sambon... ..  
 constrictor

U. S. D.

### Hæmogregarines of the Sauria

The

A number are described *H. thomsoni* Minchin 1907 in the Himalayan lizard (*Agama tubercata*) *H. schaudrini* C. Franca in *Lacerta ocellata* Daud.



Genus *Ovoplasma* De Raadt 1913

**Definition** —Toxoplasmae without definite nucleus

**Remarks** —Some doubt has been thrown upon this genus and its species

**Type Species** —*Ovoplasma anucleatum* De Raadt 1913

*Ovoplasma anucleatum* De Raadt 1913

**Definition** —*Ovoplasma* ring like with large vacuole found in man

**History** —This parasite was found in Borneo in the spleen of a

cytoplasm collects on one side of the vacuole. Sometimes it was pyriform

**Life-Cycle** —It reproduces by budding and by binary fission

**Pathogenicity** —May be harmless but in certain cases pathogenic

FAMILY *Piroplasmidae* França 1909

**Definition** —Haemosporidia without haemozoin living in red blood corpuscles

is the resultant of *Globidinium* Neumann require further investigation

gation

The following genera can be differentiated —

- 1 *Piroplasma* Patton, 1895
- 2 *Smithia* França 1909
- 3 *Nuttallia* França 1909
- 4 *Theileria* Bettencourt França and Borges 1907
- 5 *Achromaticus* Dionisi 1900
- 6 *Rangelia* Carr and Muciel 1914
- 7 *Rossella* Nuttall 1910
- 8 *Ellespsisoma*

Sellers and Gastr

burn 1915

A *Cytoplasm voluminosus*

- I Rounded forms in red cells with circular nucleus  
Schizogony by binary division inside red cells  
Division may continue and form a number of large merozoites—*Rossella*
- II Oval forms in red cells which they dehaemoglobinize  
Nucleus large at one side of parasite : Schizogony in the lung—*Ellespsisoma*

**Development in the Tick.**—When an adult tick or a nymph bites a dog and takes in blood containing the oval parasites already described, the parasites develop in the gut into round or oval bodies 4 to 5  $\mu$  in diameter, the chromatin remaining undisturbed.

clinche into two types—the acute always fatal and the subacute ending in recovery.

**Acute Form**—In the severe attack the dog quickly becomes ill with high fever ( $40^{\circ}$  C) accompanied with great weakness. After the attack of fever comes a stage of subnormal temperatures. The mucous membrane becomes

smallest amount observed being 17 per cent. Usually the leucocytes are increased up to even 60,000 but in some cases they are reduced. Polychromatophilic erythrocytes share the general increase and are frequently met with. The animal dies usually by progressive anæmia and feebleness.

times no macroscopical lesions are to be seen.

**Treatment**—Quinine benzoate of soda, calomel, etc. have all been advised. Nuttall and Hadwen have introduced Trypanblue treatment with success.

Two slight variations of the process have been described by Nuttall and

1

1

1 1 1

and occurred in the peripheral blood and in that from a kidney. They

of these parasites is not yet understood.

Free parasites with flagella like processes have been seen by Kounil, Bowhill, Le Doux, Nuttall and Graham Smith, Hanoshita, Fuleborn and Mehl and

Le Doux cells  
1906

1

1. A. Audouin in South Africa  
*Dermacentor imicola* Fabr. is suspected as the species of the disease in  
France  
Thomson 1906

This suggestion is fact confirmed by the work of  
Christophers has traced out the development in *Lutzomyia cephalis sanguinea*  
Latreille thus finally confirming the idea of the transmission through the tick



binuria, hæmaturia, and bile in the urine, and also blood in the motions. Mortality is 50 per cent.

The post mortem shows œdema of the tissues, enlargement of the spleen, inflammation of the liver, kidney, and bowels in the last of which there may be ulcers.

The *Piroplasma* is spread by the daughter adult tick developed from the *Eurhhipicephalus bursa*, which sucked the infected blood. It is inoculated into other sheep.

***Piroplasma pitheci* P H Ross, 1905**

This organism caused piroplasmosis in a species of *Cercopithecus* from Kikuyu in Uganda.

The parasite is a non pigmented pear shaped oval, or round endoparasitic body, being 1.5  $\mu$  in diameter when round and 3 by 2  $\mu$  to 2.5  $\mu$  when pear shaped.

***Piroplasma muris* Lanham, 1906**

***Piroplasma cervi* França and Borges, 1907**

This *Piroplasma* is found as bacillary and cross forms in the blood of *Cervus dama* L. Its development is not known.

***Piroplasma minense* Yakimoff, 1909**

Found in Russia in hedgehogs, and spread by *Dermatocentor reticulatus* (?)

***Piroplasma aristotelis* Denier, 1907**

This parasite is found in *Cervus aristotelis* in Annam.

**Genus *Theileria* Bettencourt, França, and Borges, 1907.**

**Synonym.**—*Lymphohæmatocytozoon* Meyer, 1913

**Definition.**—Bacilliiform or rod shaped forms arranged at times in the form of a cross.

**Type Species.**—*Theileria parva* Theiler 1903

***Theileria parva* Theiler, 1903**

**Synonyms.**—*Piroplasma theileri*, *Babesia parva* Theiler, 1903, *Lymphohæmatocytozoon parvum* Meyer 1913

This is the cause of East Coast fever in cattle in Rhodesia and is also found in India and Japan.

**Parasite.**—The parasite appears in the blood as minute bacillary forms. Very large is chromatin and lymphatic glands.

**Schizogony.**—According to Gonder the large multinucleated plasmodial masses divide into minute merozoites, and lead to the breaking up of the enclosing lymphocyte. The merozoites penetrate into another lymphocyte.

**Piroplasma gibsoni** Patton 1910

This Piroplasma has been found in dogs and in the jackal (*Canis aureus*) in India by Patton

enclosed in one leucocyte

**Pathogenicity** — It causes one variety of canine piroplasmiasis

**Piroplasma bigeminum** Smith and Kilborne 1893

**Synonyms** — *Pyrosoma bigeminum* Smith and Kilborne 1893 *Aphiosoma bigeminum* Wandollek *Babesia bovis* Clauvelot *Ixodiosplasma specificum* Schmidt

*P. bigeminum* is the cause of Texas fever in oxen and appears as pyriform round or amoeboid cells and also as flagellate forms. It can be cultivated



**Piroplasma bovis** Laveran 1888

**Synonyms** — *Babesia bovis* Laveran 1888 *Piroplasma bovis* Patton 1910

causes jaundice and anaemia. The post mortem shows haemorrhages into many organs

**Piroplasma ovis** Babès 1880

**Synonyms** — *Haematococcus ovis* Babès *Piroplasma ovis* Laveran *Imo ovis* *Piroplasma polyphagum* Bonome

*P. ovis* is found in sheep in Europe, Africa and the West Indies as large intracorpuscular and extracorpuscular forms and causes anaemia, haemoglobinuria

**Thelleria collii** Castellani and Chalmers, 1910.

This parasite is found in *Alaca us plicatus* in Ceylon, in bacillary and pear shaped forms, lying side by side in the same erythrocyte. The development has not been traced.

**Thelleria buffali** Neveu-Lemaire, 1912**Genus Nicollia** Nuttall**Nicollia quadrigemina** Nicolle, 1907.

This parasite is found in *Ctenodactylus gondii* in North Africa and Nuttall and Graham Smith point out that its method of division and its chromatin are so peculiar that its position is doubtful.

**Genus Nuttallia** França, 1909.

**Definition.**—Oval or pear shaped parasites with multiplication in the form of a cross.

**Type Species.**—*Nuttallia equi* Laveran 1899

**Nuttallia herpessidis** França 1908**Nuttallia equi** Laveran 1899

donkeys, and  
shire, in 1883,  
by Hutchison

d by the blue

th high fever,  
The animal is  
anæmia  
ular  
ighly  
e or

LOW  
chronic

- from two to five days but the  
infections may take place  
etc. stupor and anæmia of  
blood  
æmic,  
intes-  
if  
murus

breaks up into sporozoites

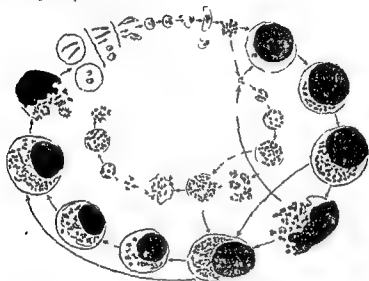


FIG. 164.—LIFE CYCLE OF *Theileria parva* (THEILER 1903)

(After Gonder from the Report of the First Expedition of the Harvard School of Tropical Medicine to South America in 1913)

It is spread by *Euryhipoccephalus appendiculatus* Meumann by the nymphs and the adults

Cultivation

*r. vicinum* but there is very little anemia and no haemoglobinuria

Mortality.—The mortality is about 90 per cent

Post-Mortem.—The autopsy shows edema of the lungs inflammation of the lymphatic glands and infarcts in the lungs liver and kidneys

*Theileria mutans* Theiler 1907

Synonym.—*Pyroplasma mutans* Theiler 1907

This *Leishmania* is found along with *P. bigemina* in cattle in the Transvaal and produces forms like *Theileria parva*, but distinguished by being inoculable. It is not known how it is spread

**Genus Rangella** Carini and Maciel 1914

**Definition**—Piroplasmidæ often in pairs with rounded oval or pyriform appearance with easily visible cytoplasm. Schizogony in endothelial cells in internal organs. Merozoites very numerous.

**Type Species**—*Rangella vitalis* Pestana 1910

**Rangella vitalis** Pestana 1910

This is the cause of a disease in dogs in Brazil called nambiavu.

**Genus Rossiella** Nuttall 1910

**Definition**—Piroplasmidæ of unusual type with voluminous cytoplasm not pigmented rounded form and rounded nucleus which is not peripherally placed. Schizogony in red cells division first into two and subsequently in more merozoites.

**Type Species**—*Rossiella rossi* Nuttall 1910

**Rossiella rossi** Nuttall 1910

Parasite of *Canis adustus* in Africa.

**Genus Elleipsisoma** França 1910

**Definition**—Piroplasmidæ of unusual type with voluminous cytoplasm not pigmented living in red blood cells which become dimæmoglobinized. Schizogony in the lungs.

**Type Species**—*Elleipsisoma thomsoni* França 1910

**Elleipsisoma thomsoni** França 1910

Parasite of *Talpa europea* and *T. caeca*.

**Genus Bartonella** Strong Tyzzer Brues Sellards and Gastriaburu 1915

**Definition**—Piroplasmidæ with rounded or rod shaped dividing forms sometimes in chains reproduction by binary division cytoplasm and chromatic substance often differentiated with difficulty. Motile. Habitat red blood corpuscles.

**Type and Only Species**—*Bartonella bacilliformis* Strong Tyzzer Brues Sellards and Gastriaburu 1915

**Bartonella bacilliformis** Strong Tyzzer Brues Sellards and Gastriaburu 1915

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paratyphoid group

in Oroya  
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pe  
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~''~ bodies in th red cells of  
In 1909 he noted these  
and stated that they were

Genus *Smithia* Franca 1910

Definition.—Coccus like parasites round or oval in form apparently consisting wholly of chromatin and devoid of cytoplasm. Flagellate forms said to exist.

Genus *Anaplasma* Theiler 1910

Definition.—Coccus like parasites round or oval in form apparently consisting wholly of chromatin and devoid of cytoplasm. Flagellate forms said to exist.

Dias and Aragão consider these organisms to be degenerations of red cells.

Type Species.—*Anaplasma marginale*

*Anaplasma marginale* Theiler 1910

Coccus like parasites round or oval in form.

Pathogenicity.—It causes a disease like red water but different therefrom in that animals immune against red water are susceptible to it. Clinically it is milder than the above form.

*Anaplasma marginale* var. *centrale* Theiler 1912

Like *A. marginale* but situate towards the centre of the cell. Type of illness milder than the above form.

*Anaplasma canis* Basile 1912

Cocci like forms and crescent shaped bodies free and enclosed in corpuscles found in peripheral blood liver spleen and lungs of dogs around Messina. Large form 4.9 by 2.3  $\mu$  observed provided with a flagellum measuring 3  $\mu$ .

Pathogenicity.—Causes canine anaplasmosis.

Genus *Achromaticus* Dionisi 1898

Definition.—*Piroplasmidae* with easily visible but not voluminous cytoplasm sickle shaped pyriform or rounded. S. hazogony in red cells. Many microzoites. Large solitary parasites.

Type Species.—*A. vesperuginis* Dionisi 1898

*Achromaticus vesperuginis* Dionisi 1898

original file of a separate genus. Recently it has been studied by Yakimoff, Stolnikoff and Kohl Yakimoff who believe that it is a true *Piroplasma*. Another species is *A. gibsoni* Patton 1910.

gested that the parasite breaks up into a large number of minute elements each of which possesses a chromatin granule. These elements grow and become rods which are set free by rupture of the enclosing sphere and so fill the endothelial cells from which they escape also by rupture. The rods are looked upon as gametocytes rather than merozoites and are considered to be the forms seen in the red cells.

**Comparison**—The organism is believed to resemble *Theileria parva*.

**Inoculations**—Attempts to transmit the parasite to inoculated animals failed. The animals used were rabbits and monkeys.

could be obtained.

**Pathogenicity**—It is believed to be the causal agent of Oroya fever.

#### FAMILY PLASMODIDÆ LUHE 1906

*sporilia* Wasie  
Minchin 1903  
the trophozoite  
grows into the schizont containing hæmozoin which breaks up into a number of merozoites which are usually said never to be flagellate. The ookinete encysts and forms a typical oocyst which breaks up eventually into sporozoites.

are *Plasmodium*  
and Feletti 1889

A Size large Schizogony binary or at times quaternary in

B or merozoites—

#### II With crescent bodies—*Laverania*

##### Genus *Plasmodium* Marchiafava and Celli 1885

**Synonyms**—*Oscillaria* Laveran 1881; *Hæmatomonas* Osler 1887; *Hæmatophyllum* Metchnikoff 1897; *Hæmatæba* Grassi and Feletti 1889; *Laverania* Grassi and Feletti 1889; *Cylamæba* Danilewski 1890; *Proteosoma* Labbé 1894; *Hæmosporium* Lewkowicz 1897

I  
fes resemble more  
hizogony in the

**Nomenclature**—Some remarks are necessary on the nomenclature. Laveran first used the term *Laverania* because he saw the flagellate form which at that time was thought to be a

protozoa and probably the cause of the disease. In the same year Gastraburu and Rebagliati confirmed these findings and regarded the organism as the cause of Oroya or Carrion's fever. In 1915 Strong Tyzzer Brues Sellards and Gastraburu confirmed and enlarged these discoveries and named the parasite.

**Morphology.**—In fresh blood films the parasite appears as a rounded or rod shaped body 0.5 x 0 microns in diameter in the former case and 1.5 x 2.5 microns in length in the latter. They are very abundant in severe cases and are endowed with a definite motility which is totally distinct from pedesis. They glide slowly about the cell. Sometimes a dot or bead like body can be seen at the two poles.

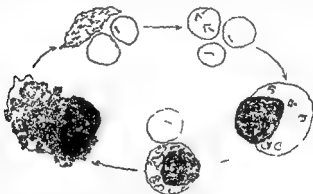


FIG. 165.—LIFE CYCLE OF *Bartonella bacilliformis* STRONG TYZZER BRUES SELLARDS AND GASTRABURU 1915

(Constructed from the illustrations in the Report of the First Expedition of the Harvard School of Tropical Medicine to South America in 1913)

When stained they are seen to be slightly curved and to be present as single individuals pairs or in chains of three to five. V and Y forms are not uncommon and are considered to represent dividing forms. Cross forms are rare and due to organisms being superimposed.

than the rest

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destroy by the bacteria in a single cell which they apparently

destroy

cells

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rod

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com

chromatin which vary in number from one to many and it is sug-



twenty to thirty sporoblasts whose periphery will be marked by a palisade of forming sporozoites

Between the fourth and fifth day the cysts ( $50 \mu$  in diameter) full of sporozoites (size  $1.4 \mu$ ) will be seen to be projecting into the coelome

After the seventh day the oocyst ruptures and the sporozoites escape and find their way to the salivary glands in the cells of which they lie mainly in those of the mid or poison gland Thus about the tenth to the twelfth day after infection the mosquito is ready to spr

is complete

infection of

into the lar

generation is not known

### *Plasmodium malariae* Laveran 1881 (Plate I)

**Synonyms**—*Hamamocba malariae* Grassi and Feletti 1890  
*H lateran* var *quartana* Labbe 1894 *H malariae* var *magna*  
 Laveran 1900 *H malariae* var *quartanae* Laveran 1901 *Plasmo-*  
*d malariae* var *quartanae* Celli and Sanfelice 1891 *Hæmospor*  
 " *quartanae* Jannesco

aller than

1 on thro

ment will be noticed to be gathered at the periphery and to be very dark in colour and non motile The red cell tends if any thing to become smaller and darker In about sixty hours the trophozoite will have become the full grown schizont which is a large round pigmented body surrounded by a rim belonging to the corpuscle During the next twelve hours its nucleus divides up into six or twelve nuclei around which the cytoplasm gathers while the hæmoglobin is driven into the centre and the appearance of a daisy is produced by the central block of hæmoglobin and the regular arrangement of the merozoites around it (size  $6 \mu$ )

The merozoites (size  $1.75 \mu$ ) are now set free and as a rule many of them appear cells More rare recently have been said a severe infection is not usual

The whole schizogony takes place in the peripheral blood and occupies seventy two hours

**Sporogony**—Gametocytes are very rarely seen and only after the disease has lasted a long time but Vida has recently described all stages of their development as seen in the peripheral blood

by Lillie and Celli in 1885

It is not a good term because a plasmodium is generally considered to be a mass of protoplasm with several nuclei representing

parasites may be taken as the

structure outside the human body in certain species of different genera of the family Anophelinae a type of mosquito which is somewhat easily identified by its

asexual bodies are called sporozoites and consist of

female and indifferent parasites or they may not the question is still undecided

This is the form

may all the old form disappear. First in the new form

the fully-grown trophozoite

full of pigment granules is called the schizont which has a subcentral nucleus. This nucleus now divides so that parasites may be seen with two three four five six up to twenty four nuclei. The cytoplasm around these nuclei segments into small bodies called merozoites each with a nucleus but in unsegmented portion

## PLATE I.

### THE MALARIAL PARASITES

*Coloured by Leishman's Stain.*

#### 1a—4a. PLASMODIUM MALARIE

- 1a Young Trophozoite
- 2a Older Trophozoite
- 3a Schizont
- 4a Sporulation

#### 1b—8b. PLASMODIUM VIVAX

- 1b Young Trophozoite
- 2b Older Trophozoite showing Amoeboid Movement
- 3b Schizont with single Chromatin Mass
- 4b Schizont with three Chromatin Masses
- 5b Sporulation
- 6b Young Sporont
- 7b Microgametocyte
- 8b Macrogametocyte

#### 1c—4c LAVERANIA MALARIE

- 1c Two Young Trophozoites
- 2c Sporulation
- 3c Macrogametocyte
- 4c Microgametocyte

They begin as small forms with a central nucleus but no vacuole and soon become pigmented the fully grown macrogametocyte

parasite

The microgametocyte shows the usual structure already described

It takes about eighteen to twenty one days to be completed after infection

Pathogenic to *Culex*

It was first found on the Black Sea littoral may also be *La. tania malariae*

*Plasmodium danilewskyi* Grassi and Feletti 1890

Synonyms—*La. tania danilewskyi* Grassi and Feletti 1890 *Hamamabys relicta* Grassi and Feletti 1890

These develop in *Culex*

*Culex* parasites have been found in the larva or second generation of



PLATE I.



1a



2a



3a



4a



1b



2b



3b



4b



5a



6a



7a



8a



1c



2c



3



4c

MALARIAL PARASITES  
(Coloured by Leishman & Stizzo)

To face page 216

**Plasmodium equi** Castellani and Chalmers 1913

Found by us in a horse in Ceylon It closely resembles *P. canis*

**Plasmodium brasilianum** Gonder and Gossler, 1908

Resembles the human quartan parasite but found in *Brachyotus calvus* in Brazil

**Plasmodium vassalli** Laveran 1905

Synonym.—*P. tassali* Simbon 1907

Found by Vassal in a squirrel—*Sciurus griseimanis*



FIG 172.—*Plasmodium canis* CASTELLANI AND CHALMERS  
1 Young gametocyte 2 Macrogametocyte 3 Microgametocyte

**Other Forms**

In Mammals.—*Plasmodium murinum* Dionisi, 1898 in *Myotis myotis*  
*P. monosoma* Vassal 1907 in *Iesperugo abramus*

In Birds.—*P. majoris* in *Favis major* *P. vaughani* Novy and MacNeal 1904  
in *Aerona migratoria*

In Lizards.—*P. diploglossi* Aragão and Neiva 1900 in *Diploglossus fasciatus*  
*P. tropiduri* Aragão and Neiva 1909 in *Tropidurus torquatus* Wied in Brazil

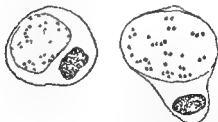
**Genus Hæmocystidium** Castellani and Willey 1904

FIG 173.—*Hæmocystidium simondi* CASTELLANI AND WILLEY  
(After Castellani and Willey)

*Hæmocystidium simondi* Castellani and Willey 1904





## DIFFERENTIAL CHARACTERS OF THE MALARIAL PARASITES

Character	<i>P. malariae</i>	<i>P. vivax</i>	<i>L. malariae</i>
Schizogony	Completion in seventy two hours	Completion in forty eight hours	Completion in forty eight hours or less
Young trophozoite	Young trophozoite smaller than <i>P. vivax</i> larger than <i>L. malariae</i> movements rather slow pseudopodia not marked or long	Young trophozoite large very actively motile long pseudopodia	Young trophozoite small actively motile
Hæmozoön	Granules coarse sluggish peripherally arranged dark brown	Granules fine movement marked	Granules fine and scanty often motionless
Schizont	Smaller than red corpuscle	Larger than red corpuscle	Much smaller than red corpuscle
Merozoites	Six to twelve regularly arranged in a rosette	Fifteen to twenty regularly arranged	Eight to fifteen arranged irregularly
Gametocytes	Resemble sporonts but larger	Resemble sporonts but larger	Crescentic in shape
Erythrocytes	Almost normal	Pale and swollen	May be small and dark

Schizogony takes from thirty six to forty eight hours to be completed

The gametocytes are characterized by being crescent shaped and large with the remains of the red cell stretched round them. The hæmoglobin of the cell is often seen lying in juxtaposition to the parasite while the remaining portion of the corpuscle is almost colourless.

The macrogametocyte is characterized by its long thin shape

## FAMILY HÆMOPROTEIDÆ Sambon 1906

**Definition.**—*Hæmosporidia* with hæmozoön but with ookinete which does not encyst

Genus *Hæmoproteus* Kruse 1890

α = *Trypano*

Feletk in  
n 1894 to  
wing that  
been sup-  
by Novy  
stake and  
In 1908

Genus *Laverania* Grassi and Feletti 1890

**Definition**—Plasmodiæ in which the gametocyte is dissimilar from the schizont appearing in the form of a crescent. Schizogony in the red blood cells in internal organs

*Laverania malariae* Grassi and Feletti 1890 (Plate I)

**Synonyms**—*Hamantzba malariae* Laveran 1890 *H. praecox* Grassi and Feletti 1890 *H. laverani* Iabbe 1894 *H. immaculata* Grassi and Feletti 1891 *Plasmodium malariae* var. *quotidiana* Cell 1893

*Hamatozoon falsiparum* Welch 1897

FIG 174—MALE CRESCENT OF *Laverania malariae* (GRASSI AND FELETTI 1890) (X 1 000 DIAMETERS)

FIG 175—FEMALE CRESCENT OF *Laverania malariae* (X 1 000 DIAMETERS)

(By Norman through the kindness of J J Bell)

The young trophozoite begins as a very small parasite which quickly forms a ring of which the size is only one-sixth to one-seventh of the diameter of the enclosing red cell. This quickly grows into an oval form which becomes pigmented while the enclosing erythrocyte in stained preparations may show the so-called Maurer's dots the significance of which is not understood.

The fully-grown schizont (size 4.5  $\mu$ ) is but rarely seen in the

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this

the form of dots at regular intervals



FIG 178 — *Hamoproteus noctua* CELLI AND SANFELICE THE OOKINETE



FIG 179 — *Hamoproteus noctua* CELLI AND SANFELICE MATURATION OF THE OOKINETE

(After Schaudinn)

The kinetonucleus is situate in the posterior third of the cytoplasm and consists of a rather elongated mass with eight chromosomes and one intra nuclear centrosome

In addition to this centrosome there are two others an anterior situate



FIG 180 — *Hamoproteus noctua* CELLI AND SANFELICE

(After Schaudinn)

Formation of the indifferent ookinete and its development into the indifferent trypanosome

• • • • •

## Hæmoproteus noctuæ Cilli and Sanfelice 1901

*H. noctuæ* goes through the cycle of schizogony in *Glaucidium noctuæ* Retz the little owl and its sporogony in *Culex pipiens* Linnæus



FIG 176—*Hæmoproteus mansoni* SAMBON SHOWING THE DEVELOPMENT OF THE GAMETOCYTES  
(After Sambon)

When this gnat sucks the blood of a little owl infected with hæmoproteus two halteridial forms in the owl's corpuscles are seen to undergo development in its stomach. These two forms are the microgametocytes and the macrogametocytes.

**Microgametocyte**—The microgametocyte appears as a typical halteridium parasite enclosed



FIG 177—*Hæmoproteus noctuæ* CELLI AND SANFELICE

(After Schaudinn)

On the left is the microgamete and on the right a scheme showing the arrangement of the nuclei centrosomes undulating

1130

The whole process has been carefully worked out by Macallum in another species of halteridium and is confirmed by Schaudinn in the present species.

**Formation of the Microgametocyte**—The microgametocyte is a clear hyaline body which on escaping from the red blood corpuscles throws out active flagella which after beating about a little break loose forming the free microgamete.

## CHAPTER XXII

# NEOSPORIDIA

Neosporidia—Myxosporidia—Actinomyxidia—Sarcosporidia—Haplosporidia  
—Protozoa incertae sedis—Chlamydozoa—Filterable viruses—References

### NEOSPORIDIA Schaudinn 1900

**Definition**—Parasitic plasmodiomata without motile organs in which spore formation

**Remarks**—The  
and growth go on  
grows into the schizont which divides into spores in the Neosporidia

dinal ancestor

They are divided into four orders (1) Myxosporidia (2) Actinomyxidia (3) Sarcosporidia (4) Haplosporidia and in addition there are a number of parasites belonging evidently to the protozoa which cannot easily be classified and are therefore placed in an addendum to the Neosporidia as *Protozoa incertae sedis*

### ORDER I MYXOSPORIDIA Butschli 1881

sub. 1. sub.

by treatment with reagents

### SUBORDER I PHAENOCYSTES Gurley 1893

Phaenocystes comprise the true Myxosporidia being usually found in Teleostean fish though they may occur in elasmobranchs, amphibia and reptiles

r. b. f. d. n. h. b. l. e

reduction which results in four dyads and then a second division follows leaving now retinas and the micro

the kinetonucleus but it ultimately consists of four chromosomes and a centrosome which Woodcock points out are not wholly sexual



FIG 181—*Haemoproteus noctua* CELLS AND SPINDLE (After Schaudinn)

Formation of the male ookinete and its development into the male trypanosome

**Zygote**—This consists in the penetration of a microgamete into a receptive cone which has arisen from the cytoplasm of the macrogamete on the side where the nuclei are situated

The only parts which enter are the male trophonucleus which is reduced and the male kinetonucleus which is not reduced but which now undergoes two divisions. The trophonuclei of the male and female elements fuse and form the fusion spindle at either end of which the kinetonuclei take up positions and thus the zygote is formed

*Ookinete* etc.



FIG 182 *Haemoproteus noctua* CELLS AND SPINDLE DEVELOPMENT OF THE FEMALE OOKINETE AND THE FEMALE TRYPANOSOME

(After Schaudinn)

eight chromosomes with a centrosome in the center

retains consists of a central round which lies at other

Schaudinn describes three

183—

**Indifferent Ookinete**—Cytoplasmic and staining faintly with one or two large vacuoles anteriorly and having some granular material and hemazon still left



pansporoblast The nucleus of the pansporoblast divides repeatedly after which the cytoplasm splits into two masses—the sporoblasts—each of which

### SUBORDER II CRYPTOCYSTES

This order is divided by Doëlein and Perez into —

- Tribe 1 *Monosporogenea* Perez — Trophozoite becomes a single pansporoblast (larva) which produces many spores
- Tribe 2 *Oligosporogenea* Doëlein — Trophozoite becomes a single pansporoblast (larva) which produces many spores
- Tribe 3 *Polysporogenea* Doëlein — Trophozoite becomes a single pansporoblast (larva) which produces many spores

#### TRIBE 1 MONOSPOROGENEA

This tribe includes *Nosema bombycis* Nageli 1857 which is the cause of pébrine the silkworm disease. *N. apis* Zander 1909 was shown to be the cause of microsporidiosis in bees in England by Fantham and Porter

#### TRIBE 2 OLIGOSPOROGENEA DOËLEIN 1899

This includes the genera *Gurleya* Doëlein *Thelohania* Henneguy and *Pleistophora* Gurley

#### TRIBE 3 POLYSPOROGENEA DOËLEIN 1899

This includes the genera of *Glugea* Thelohan and *Mycosporites* Mrazek of which *Glugea anomala* Moniez is a parasite of the sea kelpback. *Microsporidium foledricum* Bolle a doubtful species is said by Ferronci to occur in man

### ORDER II. ACTINOMYXIDIA Stoll 1899

These are parasites in the Tubificidae of the oligochaete worms and need not concern us



gested that the parasite may be transmitted by the larvæ and imagines of the blow fly (*Calliphora*) or the flesh fly (*Sarcophaga*)

It is true that Smith has infected (after a long incubation) mice by feeding them on the flesh of infected mice but that of course might simply be by the merozoites and would in any case not explain how herbivorous animals are infected. Our feeding experiments with a dog were not successful

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where it lives

Crawley considers that the spore bores its way into the intestinal cells where it appears to undergo some form of schizogony. At all events it disappears in twenty-four hours but later he thought that he had noted sexual differentiation in these spores in the cells

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They were first described indefinitely by Lindemann in 1868 in the myocardium of a man who had died of dropsy, 5 millimetres in breadth.

of a cyst in a papillary  
endocarditis Kartulis

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contained only gymnosporous (merozoites) and no pansporoblasts or alveolar network. The spores contained nothing but a nucleus, no cell membrane or other structure being visible. There were no signs of any reaction on the part of the tissues.

parasite found by Korté  
that the inner coat was  
ite and the endoplasm

while farther in chambers with the fully-developed pansporoblasts are found and still farther in is the centre of the parasite filled by a granular substance formed from broken down and dead spores



FIG 188 — *Sarcocystis tenella* cysts IN MEAT

some granules and one or two vacuoles. They are



FIG 89 SARCOCYSTIS SPORES FOUND IN THE BLOOD OF DOGS WITH BY CASTELLANI AND STURGESS

thought to be the means by which the parasite spreads itself in its host especially as they are motile by gliding corkscrew or amoeboid movements

The spores are from 3 to 14  $\mu$  long in *Sarcocystis tenella* with one extremity rounded and the other pointed. They are curved and surrounded by a thin membrane. The pointed third of the spore is spirally striated due to fine folds in the outer capsule while the blunt third contains a nucleus. It is obvious that this resembles the spore of a myxosporidian and in fact Van Eecke says that one two or even three filaments issue from a spore. The spores can be seen in the peripheral blood at times.

It is thought that these spores spread the infection to another host but in what manner is not clear. Ferrin has recently shown

gested that the parasite may be transmitted by the larvæ and  
 (ophaga)  
 (ation) mice  
 of course,  
 might simply be by the merozoites, and would in any case not  
 explain how herbivorous animals are infected. Our feeding experi-  
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 They were first described indefinitely by Landemann in 1868 in the myo-  
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 not contained any merozoites (merozoites) and no pansporoblasts or alveolar  
 network. The spores contained nothing but a nucleus, no cell membrane  
 or other structure being visible. There were no signs of any reaction on the  
 part of the tissues.



myxomatous in places and cellular at other spots. The cysts were lined or irregular bodies lined but also found in hæmorrhages. The cyst is generally thin and has either an opening or a conical elevation at one point. It consists of two layers—a thinner external and thicker internal coat. The smaller cysts from 10 to 30  $\mu$  in diameter contained undifferentiated protoplasm with a vesicular nucleus containing a nucleolus. The larger cysts had one or more definite chromatic masses. A fully developed cyst is lined with protoplasm in which young pansporoblasts are forming while the centre of the parasite is full of old pansporoblasts separated from one another by an indefinite framework continuous with the capsule. A young pansporoblast is seen to be a small oval or rounded mass of cytoplasm with a single nucleus. This body grows and becomes surrounded by a membrane while its nucleus divides by amitosis into four to sixteen spores each of which has a very thin wall and a central nucleus. The pansporoblasts and spores are set free by rupture of the cyst and may be surrounded by polymorphonuclear leucocytes thus forming minute abscesses or may be engulfed by mononuclear leucocytes or may grow into parasites or escape from the host in the nasal secretion. The method of infection is not known.

The framework inside the cyst separating the pansporoblasts indicates that *Rhinosporidium* belongs to the Sarcosporidia and not to the Haplosporidia.

**Pathogenicity**—The pathology appears to be a proliferation of the submucosa and mucosa of the nose brought about by the irritation of the parasite (see p 1578).

#### ORDER IV HAPLOSPORIDIA Caullery and Mesnil 1899

**Synonym**—*Haplosporididea* Poche 1913

Neosporidia with very simple life history and undifferentiated cell plasma without septum and with spores of simple structure with one nucleus and no polar capsules.

is unknown

1—Spores

annelids

**FAMILY 2 BRYTRAMIDÆ**—Spore envelope without opening

Genus 1 *Bertramia*—Stomach of fish

Genus 2 *Ichthyosporidium*—In tumours of fish (Figs 191 196)

**FAMILY 3 COLLOSPORIDIIDÆ**—Spores nude

Genus 1 *Polagarynum*

Genus 2 *Blasididium*

8 *S. aramidis* Splendore 1907—Parasite in *Aramid's azrae* in a Brazil bird

9 *S. ammodromi* Splendore 1907—*Miescheria ammodromi* Splendore 1907  
Merial says it is not generic, and perhaps not specific. It is found in a Brazil bird *Ammodromus pinnatus*

10 *S. leporum* Crawley 1914 in American rabbit

11 *S. setophaga* Crawley 1914 in American redstarts

12 *S. muris* Blanchard in rats

## 2 Rhinosporidiidae Poche 1913

**Definition**—Sarcosporidia found in connective tissue and not divided into chambers by septa

### Rhinosporidium Minchin and Fantham 1905

**Definition**—Rhinosporidiidae with well defined sporoblast

**Type Species**—*Rhinosporidium seeberi* (Wernicke 1901)

### Rhinosporidium seeberi Wernicke 1900

**Synonym**—*Rhinosporidium kincaelyi* Minchin and Fantham 1905  
*Rhinosporidium* was discovered in 1896 by Dr. Guillermo Seeber  
1917  
and in

Laryngological Society a peculiar



FIG. 190.—*Rhinosporidium seeberi* WERNICKE

(From a nasal polypus in a case in Cochin schematic and highly magnified)

section this tumour was found to have peculiar bodies embedded in it

It was then carefully examined and described by Minchin and Fantham who came to the conclusion that it was a haplosporidium and named it *Rhinosporidium kincaelyi*

In 1905 Naik of Madras came across a similar polypus in several people who all came from the small native State of Cochin on the west coast of India. These polypi have been carefully described by Britton in 1906. Castellani



FIG 191 — A SERIES OF SMALL ICHTHYOSPORIDIA ENCLOSED IN CONNECTIVE TISSUE



FIG 192 — A MEDIUM SIZED ICHTHYOSPORIDIUM IN A NEST OF CONNECTIVE TISSUE

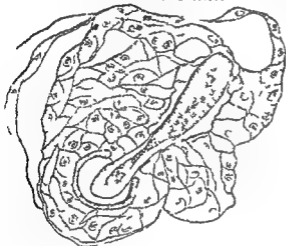


FIG 193 — ESCAPE OF THE ICHTHYOSPORIDIUM FROM ITS CYST



FIG 194 — BREAKING UP OF AN ICHTHYOSPORIDIUM INTO REPRODUCTIVE BODIES

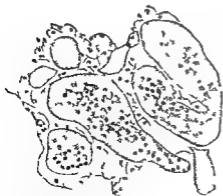


FIG 195 — LARGE ICHTHYOSPORIDIA ONE OF WHICH (ON THE RIGHT) IS UNDER GOING PLASMOTOMY



FIG 196 — YOUNG FORMS DEVELOPED FROM THE BREAKING UP OF A LARGE PARASITE

(From drawings by Miss Robertson)

Protozoa Incertæ Sedis

*Cytoryctes varifolus* Garneri 1892

plasm form a *nucleus de reliquat*

**Sexual Development**—The homogeneous granules or gemmules may start the cycle of cytoplasmic organisms again or may become germ-cells in the nucleus. The gemmules reach the nucleus but they now stand unformly

**Cytoryctes (Doubtful Species)**

This organism can be found as corroded bodies in the smears taken from the heart muscle of animals suffering from foot and mouth disease

*Neurocyctes hydrophobus* Calkins 1907

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It is there cannot be any doubt that they are parasites especially after the f the London



Bodies found by Sambon in *Pseudochirus peregrinus*

Sambon has found bodies in a lemur which may have some relationship to the spores of *Sarcocystis* but this is doubtful

## Bodies described in Man by Castellani and Willey.

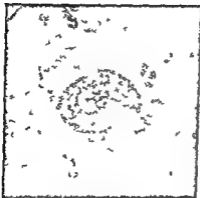


FIG 199 — *Entoplasma Castellani*  
PAUL, 1914

cellularly, when they are capable of passing through the usual  
" " " " when they excite a reaction upon the

embrace a number of minute particles  
closed in a cellular product as with a mantle The general  
tendency is to range these forms among the Protozoa The minute  
granules are the parasites and the surrounding substance is either  
" " " " from the nucleus or a fatty substance The

trachoma smears stained by Giemsa's method

appeared as large pear shaped or flask  
shaped bodies 60 80 microns in diam  
showing only

seen in fresh or stained preparations  
by Castellani or any other observer  
The cytoplasm was very vacuolated  
In stained preparations a group of  
granular bodies could be seen and were  
thought by Mesnil to be a diffuse nucleus

*Chlamydozoa* Prowazek 1907

**Definition.**—The *Chlamydozoa*  
are a collective group of minute  
parasites which either live extra  
cellularly, when they excite a reaction upon the

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**Cyclasterella scarlatinalis Mallory (Doubtful Species).**

In 1904 Mallory described -

Further research is needed before the nature of these bodies can be definitely settled



FIG 197 — *Sergentella hominis*  
BRUMPT.



FIG 198 — SARCOCYSTIS SPORES  
FOUND IN THE BLOOD OF MAN BY  
CASTELLANI AND WILLEY

**Coccidioides immitis Rixford and Gilchrist 1897 (A Fungus)**

Synonyms — *C. posadasii* Rixford and Gilchrist 1897 *Coccidium posadesii* Caxton (?) 1898

These parasites are found in the lungs and later in the spleen and Ophiostoma in the liver and kidneys. This so-called

**The Bodies of Ureteritis Cystica (Doubtful).**

In this disease the kidney is hydronephrotic and the ureter and bladder are cystic.

The cysts contain large and small oval and irregular cells with bright globules variously interpreted as Coccidia, Myxosporidia and cell inclusions.

**Sergentella hominis Brumpt 1910**

It and Ed. Sergent in 1903 reported a vermiform body 40  $\mu$  long by 1 to 5  $\mu$  broad pointed at each end with a nucleus in the middle in the blood of a person suffering from night sweats and paræcia.

## CHAPTER XXIII

# HETEROKARYOTA

Preliminary—Heterokaryota—Ciliata—Balantidium—Nyctotherus—References

### DIVISION B HETEROKARYOTA HICKSON 1903

#### PHYLUM V CILIATA Perty 1852

THE Ciliata are free-living or parasitic Heterokaryota found principally in water where they exist upon small animal and vegetal organisms and the debris of decomposing plants and animals. Some of them can live in the alimentary canal of man and animals obtaining their food from its contents and increasing

the posterior or may be characterized by being more pointed by having a mouth or by peculiar sensory cilia (Hypotricha) or by a peristome of long cilia (Heterotricha). The body which may be spherical or flattened is divided into an ectoplasm (the cortex) and endoplasm (the medulla). The ectoplasm may simply be a clear outer layer of the protoplasm or consist of two or more layers. The first is very thick and is marked by the alveolar sheath is marked by the contractile myoneme threads while the innermost layer next to the endoplasm consists of clear transparent ectoplasm.

The semifluid endoplasm is in constant rotatory motion containing food vacuoles, contractile vacuoles, nuclei, pigment granules, and smaller particles. The contractile vacuole is usually located at the anterior end of the body which can be on the surface or may be carried inwards by a funnel-shaped depression in the ectoplasm called the vestibule which may be

through the cortex

granular inclusions in the protoplasm of the epithelial cells. These granules were at first round or oval and increased in size at the same time becoming less dark while minute red dots appeared which increased rapidly in numbers while the blue masses gradually disappeared. The granules formed cell inclusions and the blue masses were considered to be a reaction product on the part of the cell and were thought to be composed of plastin while the minute red dots

reason why they considered the red points was because the red points or elementary situated. They inoculated

anthropoid apes successfully with trachoma and found the same bodies in this infection. These researches were confirmed by Greef in the same year and were extended in 1905 by Stürgardt and Schmeidler in 1909 who described a conjunctivitis neonatorum non gonorrhoeica with typical Chlamydozoa. In 1909 Heymann found the same bodies in four cases of gonorrhoeal conjunctivitis in newly born infants. This discovery was of the greatest importance because since the days of Kroner it had been known that the conjunctivitis of the new born was not always due to the gonococcus. Linder in 1909 and Wolfram in 1910 showed that there were two forms of blennorrhoea—viz a conjunctivitis neonatorum caused by the gonococcus and a second caused by Chlamydozoa this latter disease being termed by Linder inclusion blennorrhoea in contradistinction to gonoblennorrhoea. Linder maintains that the same virus produced trachoma inclusion blennorrhoea and that this virus can be found in the male and female genital passages and he bases his opinion on the facts that he has been able to produce trachoma in monkeys inoculated from a case of non gonorrhoeal urethritis in a man from two such cases in women and from several cases of inclusion blennorrhoea in infants. Further investigations have shown that inclusion blennorrhoea is histologically similar to trachoma.

Later Ieber and Prowazek in 1911 found a similar organism *Chlamydozoa atrophicans* in epitheliosis desquamativa and in the same year Uhlenhuth found inclusions in swine pox and Botteri in spinous catarrh.

In the meanwhile Halbräedter and Prowazek had in their first paper grouped with these cell inclusions the forms described in

four micro and four-macro nuclei are in the same cell which divides into two very small cells with two macro and micro nuclei each. These cells grow to nearly full size and then divide giving rise to the typical protozoon with one macronucleus and one micronucleus.

**Parasitism.**—A great many species of the Ciliata are parasitic in the intestine or bladder of other animals and some are epizootic.

The latter will often be met with as *Vorticellæ* living on *Anopheles* and *Culex* larvae. The former are found largely in the Orthoptera, the Amphibia and in herbivorous mammals—e.g. horse and cow.

In man a few have been recorded. *Chilodon dentatus* Dujardin 1842. *C. uncinatus* Colpoda *cucullus* Schutz 1889. *Balantidium coli* Malmsten, 1857. *B. minutum* Jakob and Schaudinn 1898. *Nyctotherus faba* Jakob and Schindinn 1898. *N. giganteus* Krause 1906. *N. africanus* Castellani 1905.

Parasitism does not appear to affect the structure of the animal unless the loss of the cytostome in *Opalina* parasitic in frogs is considered to be due to this cause.

**Pathogenicity.**—The ciliate parasites as a rule appear to cause but little effect unless they are present in large numbers when diarrhoea often severe and long persisting may result.

**Classification.**—The Ciliata are classified into four orders—

**Order I Holotricha** Stein 1859—Mobile Ciliata without special oral cirri (*Chilodon* *Colpoda*)

**Order II Heterotricha** Stein 1859—Mobile Ciliata with special oral cirri (*Balantidium* *Nyctotherus*)

**Order III Hypotricha** Stein 1859—Mobile Ciliata with well developed dorsal and ventral surfaces. Not known to be parasitic in man.

**Order IV Peritricha** Stein 1859—Fixed Ciliata. Not known to be parasitic in man.

### ORDER I HOLOTRICHA Stein 1859

**Definition.**—The Holotricha are free moving Ciliata in which all the cilia are of approximately equal length and thickness and

food  
divided

with an undulating membrane

#### SUBORDER GYMNOSTOMATA Bdtschli 1889

In this family there is a species with cilia of equal length and thickness and a strongly dorso-ventrally flattened body commonly found in infusions of which one species *C. dentatus* Dujardin 1842 was found in great abundance by Guart in the motions of a woman suffering from severe dysentery in Paris. Manson and Sambon have described a case of chance parasitism due to another species *C. uncinatus* Blochmann in a patient from tropical South Africa.

Two wholly distinct nuclei exist in the Heterokaryota a mega or macro nucleus and a micronucleus. They are not merely different in size form structure and appearance but also in function for the macronucleus is somatic and trophic in its function while the micronucleus is purely sexual. They are not comparable to the tropho- and kinezo-nuclei of the trypanosomes.

The macronucleus is generally well marked but breaks down into granules before or after conjugation. It consists of an achromatic portion with a chromatic portion in the form of a close-meshed network of fibrils but whether there is a definite surrounding membrane or not is doubtful.

The micronucleus when at rest is a minute irregular granule of chromatin lying in the centre of a perfectly clear achromatic area. Probably there is only one in each animal and the appearance of two or more is due to reproductive phases which are just finishing.

The Ciliata are characterized by the presence of protoplasmic processes projecting all over the body. These are fine short whips called cilia which in places are transformed into thick processes called cirri or flat membranes in certain species. The cilia are processes of the pellicle but they appear to be supported by a thread of specialized ectoplasm. The short fine cilia are for motion and the long motionless cirri for the purpose of entangling food particles. The membranes are supposed to be formed of fused cirri. Trichocysts exist in the Holotricha and an excretory organ has recently been described by Metcalf in certain species of *Opalina* parasitic in frogs while other Ciliata have a contractile vacuole.

Reproduction may take place asexually by (1) transverse or longitudinal division (2) gemmation simple or multiple (3) encystment and spore formation or sexually by conjugation.

**1 Fission**—There is no morphological distinction between longitudinal and transverse fission in which the cell wall changes take place. (1) A second mouth is formed then comes (2) enlargement and division of the micronucleus followed by (3) enlargement and division of the macronucleus and finally (4) division of the cytoplasm.

**2 Encystment and Spore-Formation**—The animal encysts and

The micronucleus swells its chromatin becomes granular elongated crescentic fusiform and finally forms two nuclei each of which after two or three divisions forms four nuclei and then the organisms separate. The macronucleus fragments and disappears and the synkaryon breaks up into eight micronuclei.

Four of these swell and are changed into macronuclei so that

It develops asexually  
It can encyst and in the  
lives in the rectum of pig

and is transferred by its cysts to man

It was discovered by Malmsten in a man who had had cholera and was suffering from diarrhoea and ulcer of the rectum. The ulcer had nothing to do with the disease as it healed while the diarrhoea was probably due to the parasites.

Casagrandi and Barbagallo produced catarrhal enterocolitis in

inner the enterocolitis

### *Balantidium minutum* Schaudinn 1899

The body is shortly oval with a pointed anterior extremity.  
Length 20 to 32  $\mu$  breadth, 14 to 20  $\mu$

The peristome extends into the centre of the body and has the right lateral border fringed with cilia and the left lateral border

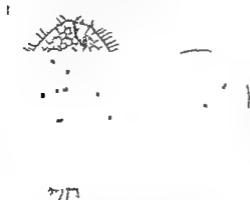


FIG 200—*Balantidium coli* MALMSTEN  
(After Hartmann From the *Archiv f. r. Schiff-  
u. Tropenhygiene*)

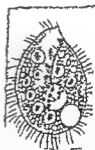


FIG 201—*Balantidium minutum* SCHAUDINN  
(After Hartmann From the *Archiv für Schiff-  
u. Tropenhygiene*)

terminates in a hyaline membrane which can pass over to the right side and has a row of cilia. The cysts are oval.

This parasite (along with *Nyctotherus fava*) was found by Schaudinn in a German who had often stayed in North America. The symptoms were constipation alternating with diarrhoea associated with abdominal pain.

### *Balantidium minutum* var. *italicum* Sangiorgi and Ugdulena 1917

This parasite which was found by Sangiorgi and Ugdulena in human faeces differs from *B. minutum* in that the nucleus is eccentric and in the peculiar orientation of the micronucleus.

They noted that the parasites were only found in the mucus never in the faecal masses. They were present in very large numbers and were found to be all gnetes some of which were conjugating. It appears probable that Guart's parasites may really have been *C. uncinatus* and not *C. dentatus*.

#### SUBORDER HYMENOSTOMATA Hickson 1913

**FAMILY CILIFERIDÆ Butschli**—The genus *Colpoda* Muller, 1773 includes the kidney-shaped Chilifera with rows of cilia twisted from left to right commonly found in hay infusions of which one species *Colpoda cucullus* Schutz 1899 commonly found in the water of marshes was noticed in a brickmaker attacked with dysentery in Berlin. *Uronema caudatum* Dujardin 1841 has been found in the motions of cases of diarrhoea in man.

#### ORDER II HETEROTRICHA Stein 1859

The Heterotricha are free-moving Ciliata with strong cilia or membranellæ forming an adoral rim enclosing a space the peristome at one part of which the mouth opens. The order is divided into Polytricha Oligotricha

*Polytricha* Habronema 1859 17 + not ab = 1 1 the surface of man *Bursaria*

#### FAMILY BURSARIIDÆ Kent 1850

**Synonym**—*Bursariidæ* Butschli

This family includes the genus *Balantidium*

#### Genus *Balantidium* (Laparede and Lachmann 1855)

**Definition**—*Bursariidæ* with a large peristome and a well marked anal aperture

**Remarks**—*Balantidium* is common in the rectum of pigs and is sometimes found in the intestines of man. Cyst 50 to 100  $\mu$  in diameter

#### *Balantidium coli* Malmsten 1857

**Synonym**—" " " " " " " "

in the colon and the dejecta of man in cases of cholera and diarrhoea. In shape it is oval slightly pointed anteriorly but this depends upon whether the peristome is funnel shaped or contracted. It is covered with cilia arranged in parallel rows which give it a striated appearance.

It is 0.06 to 0.1 millimetre in length by 0.05 to 0.07 millimetre in breadth and possesses a bean or kidney shaped macronucleus and



The macronucleus is large and bean shaped while the micronucleus is small and round. One or two vacuoles can be seen. The cytopyge is situated posteriorly.

In the feces the parasite becomes rounded off and encysts and then divides into four.

### *Myctotherus africanus* Castellani 1905

Found by Castellani in a Baganda native. *N. africanus* is hour glass shaped with the anterior portion much less developed than the posterior. Length 40 to 50  $\mu$  breadth 30 to 40  $\mu$ . The surface is covered with very minute cilia which are generally more evident on the posterior portion being almost invisible on the anterior.

The peristome is short. The cytoplasm is finely granular throughout. The nucleus is situated far posteriorly near the contractile vacuole. The micronucleus is very small and situated close to the macronucleus. No food vacuoles are to be seen.

Neither division conjugation nor encystment are known.

The patient who had diarrhoea alternately with long periods of constipation was suffering from sleeping sickness.

The cecum contained many parasites as did other parts of the large intestine. The eggs were slightly considered though not more completely.

Investigations will probably show that it constitutes a new genus.

### SUBORDER HYMENOSTOMATA Butschli 1899

#### Genus *Uronema* Dujardin 1841

**Definition**—Hymenostomata ovate or elongate with one or more caudal setae.

**Type Species**—*Uronema marinum* Dujardin 1841

#### *Uronema caudatum*

Found in a case of diarrhoea.

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CALKINS AND CAULLERY Archiv f Protistenkunde 1907 vol x 2 pp 131-137 p 375

METCALFE Arch f Protistenkunde 1907 vol x 2 and 3 pp 183-365

#### *Chilodon dentatus*

GUIART M J (1903) Comptes Rendus des Séances de la Société de Biologie

#### *Balantidium coli*

BENSEN (1908) Archiv f Schiffs u Tropenhygiene xii 672

Genus *Nyctotherus* Leidy 1849

**Definition**—Body bean or kidney shaped with a large peristome on the concave side extending from the anterior end up to the middle of the body from where a curved cytopharynx or œsophagus extends inwards. The macronucleus is large and situated almost in the centre.

**Remarks**—The species are mostly parasitic in the intestine of Amphibia Insecta and Myriapoda.

Species known in man *Nyctotherus faba* Schaudinn 1899  
*N. giganteus* Krause 1906 & *africanus* Castellani 1905

*Nyctotherus faba* Schaudinn 1899

*N. faba* is flattened dorso-ventrally and is 6 to 28  $\mu$  in length and 16 to 28  $\mu$  in breadth and 12  $\mu$  in thickness. The cilia on the peristome are of two kinds those on the right side of the size of the body being true cilia and those on the left side being cilli.



FIG. 202.—*Nyctotherus faba*  
 SCHAUDINN

(After Hartmann. From the Archiv für Schiffs- & Tropenhygiene.)



FIG. 203. *Nyctotherus africanus*  
 CASTELLANI

The contractile vacuole is large and situated posteriorly. The macular inclusions, four or six in number. The micro-

This species was discovered by Schaudinn in the same patient as *Balantidium minutum*.

*Nyctotherus giganteus* P. Krause 1906

**Synonym**—*Balantidium giganteum* P. Krause 1906

This organism along with *Erichomonas intestinalis* was found by Krause in the dejecta of persons suffering from typhoid in Breslau. It is shaped like a truncated cone with the anterior end narrowed and the posterior broad. Length 90 to 400  $\mu$  breadth 60 to 150  $\mu$ . Surface covered with cilia. The peristome is situated laterally and from it a cytopharynx leads inwards.

## CHAPTER XXIV

# TREMATODA

Metazoan parasites—Platyhelminia—Trematoda—Classification—Malacotylia—Digenea—Prostomata—Paramphistomoidea—Fascioloidea—Schistosomida—References

### SUBKINGDOM II METAZOA.

**Definition.**—Metazoa are free-living or parasitic, multicellular animals, characterized by a physiological division of labour among their cells

**Remarks.**—Tropical medicine is only concerned with parasitic Metazoa, and chiefly with those which affect man. Parasitic Metazoa may be ectoparasites—as, for example, many species of the Insecta— or endoparasites—as, for example, many worms. The ectoparasites can cause disease by introducing toxins, protozoa, or bacteria into the tissues, and in this way they are the spreaders of disease, for their range is very often limited by the animal which spreads its germs. As mosquitoes, are temporary, like lice, are permanent parasites.

With regard to the endoparasites, their ill effects on the host depend upon many factors which have been recently studied in considerable detail.

The effects of metazoan parasites on their hosts depend upon the species of the parasites, their condition, the number present, their presence in certain organs, bacterial infection, their migration in the body, the loss to the host in feeding them, the damage caused by their

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exist in a host without  
duce anæmia, œdema

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HEMUS FINGER

in the lungs will cause a disease somewhat resembling phthisis

**Balanitidium minutum**BENSEN 1908) *Op cit* 673**Nyctotherus faba.**JAKOBY AND SCHALDIN<sup>Y</sup> Centralblatt für Bakteriol u Parasitol I xxxi  
1899 487**Nyctotherus giganteus**

KRAUSE P Deutsche Archiv für Klin Med lxxxvi 442

**Nyctotherus africanus,**

CASTELLANI Centralblatt für Bakteriol Parasitol 1905 xxxviii 67 67

The old idea that worms were good for children has died out long ago and we know of no cases of mutualism or benefit to parasite and host in the animal parasites of man

True parasitism is found in those cases in which the parasite benefits and the host is injured. Chance parasites are animals which accidentally and temporarily become parasites.

The life-history of a parasite may be simple being carried out in one host or it may be complex with one or more hosts for its larval stages and another for its adult condition.

Generally these hosts bear a direct relationship to one another

eating the herbivorous host of the larva

Some parasites particularly the Microfilariae in the blood show a remarkable periodicity in their habits which appears to be associated with the means of escape from the definitive host by some or a tick whose habits agree

Such correlations are very the opening or emitting of

strong odours by certain flowers at definite times of the day or night which accords with the habits of insects which help on their fertilization

Tropical countries are the home *par excellence* for parasites as the means of infection by bad sanitation biting flies etc are easily available. It is therefore obvious that as there may be many methods of infection by the mouth the skin and the nose prevention is not an easy matter. It depends upon two factors—personal and public hygiene. The question of personal hygiene is the more important and more easily applied as only one person is concerned while that of public hygiene is more expensive and not so easy to apply as many people have to act in unison in order to produce any effect

- Phylum I Platyhelmin
- Phylum II Nematelmin
- Phylum III Annulata
- Phylum IV Arthropoda



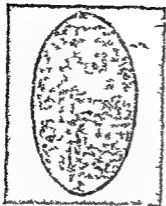


FIG 204 — *Fasciola hepatica*  
( $\times 250$ )



FIG 205 — *Fasciola buschii*  
( $\times 240$ )



FIG 206 — *Opisthorchis stenens*  
( $\times 250$ )



FIG 207 — *Schistosoma japonicum*  
( $\times 250$ )



FIG 208 — *Schistosoma kamaoebae*  
( $\times 350$ )



FIG 209 — *Schistosoma mansoni*  
( $\times 350$ )

FIGS 204-209 — EGGS OF VARIOUS TREMATODES FOUND IN HUMAN FECES

## PHYLUM I PLATYHELMIA Vogt 1871

Synonyms — *Platodes* Leuckart 1854 *Platyhelminthes* Gegenbaur

subcuticular layer in the parasitic Trematoda and Cestoda. A well developed musculo-dermal layer is present but there is no coelom.

The mouth is generally situated at the anterior end when present but it may be moved to the inferior surface and opens via a pharynx into a forked or branched gut which has no anal aperture. The alimentary canal may however be wanting (Cestoda). The excretory system begins in the so-called flame cells—flame cells provided with a leash of cilia from which fine channels run uniting together to form larger channels. Flies ultimately empty into a pair of laterally placed canals opening to the exterior separately or together often through an excretory vesicle.

The nervous system consists of a large bilobed cerebral ganglion with nerves running forwards and backwards.

The Platyhelminths are mostly hermaphrodite but may rarely be unisexual the ova are produced in the ovary near which they are fertilized and then after obtaining food yolk from the yolk reservoir which has received it via the vitelline ducts from the yolk glands they acquire a shell in a shell gland and then enter a uterus through which they slowly pass to the exterior.

The male organs consist of testes, vasa deferentia, vesicula seminalis and a cirrus pouch with a cirrus and a so-called prostate gland.

canal cuticle non-ciliate

Class III *Cestoidea* — Parasitic flat worms without an alimentary

canal cuticle non-ciliate

Class I does not enter into the subject under discussion

## CLASS II TREMATODA RUDOLPHI 1808

Synonyms — Sucking Worm

Definition — The Trematoda are parasitic Platyhelminths which retain the mouth and alimentary canal but in which the epidermis



or short pear shaped or Y shaped generally opens posteriorly but may open dorsally above the acetabulum

The sexes are but rarely separate, hermaphroditism being usual. The male organs consist of testes which may be simple or branched and are as a rule situated posteriorly. The vas deferens leads forwards, sometimes through a vesicula seminalis to the genital opening below which a cirrus enclosed in a muscular pouch provided with glands called the 'prostate' may be found.

The female ...

**Life-History.**—The full life history of a number of forms has been worked out by Leuckart and Thomas for *Fasciola hepatica* under which heading details will be given by Looss for various amphistomes, by Leiper for the genus *Schistosoma*, and by numerous

where one sexual  
s Two hosts are

required for the whole life cycle

Leiper has given the following scheme for the development of a digenetic trematode —

1	Definitive host	Egg			
2	First transition	Miracidium			
3	Intermediate host	<table border="0"> <tr> <td rowspan="4">           Sporocyst            Sporocyst and daughter cysts            Sporocyst and Rediæ            Sporocyst Rediæ and daughter Rediæ         </td> <td rowspan="4">           }            }            }            }         </td> <td rowspan="4"> <i>Cercariæ</i> </td> </tr> <tr> </tr> <tr> </tr> <tr> </tr> </table>	Sporocyst Sporocyst and daughter cysts Sporocyst and Rediæ Sporocyst Rediæ and daughter Rediæ	} } } }	<i>Cercariæ</i>
Sporocyst Sporocyst and daughter cysts Sporocyst and Rediæ Sporocyst Rediæ and daughter Rediæ	} } } }	<i>Cercariæ</i>			
4	Second transition	Free-swimming or encysted <i>Cercariæ</i>			
5	Definitive host	Adults			

Luhe has provided the following classification of *Cercariæ* (slightly modified for convenience of reference) —

A Body without internal differentiation With cuticular alveoli —  
*Lophocercariæ*

B Body with internal differentiation Tails may or may not be forked —

I Acetabulum absent—*Monostomes*

II Acetabulum present —

(a) Posteriorly situate—*Amphistomes*

(b) Ventrally situate —

1 Mouth central—*Gasterostomes*

2 Mouth terminal—*Distomes*

Jehan de Brie discovered the liver fluke in the sheep which was subsequently described by Gabucinus in 1547, but it was not till the

many observers among whom may be mentioned Lurcr (whose



FIG 210—DIAGRAM OF A TYPE OF THE FEMALE GENERATIVE APPARATUS OF A TREMATODE

(After Stiles)

1 Ovary 2 uterus 3 shell gland  
4 outyoe 5 ovarian duct 6 Laurer's canal 7 vitellarian duct

canal still bears his name) in 1630 van Beneden in 1838 and Leuckart in 1867 (who divided them into *Distomca* and *Poly-stomea*) and Thomas in 1883



FIG 211 DIAGRAM OF ANOTHER TYPE OF THE FEMALE GENERATIVE APPARATUS OF A TREMATODE

1 Ovary 2 ovarian duct 3 shell gland 4 outyoe 5 uterus 6 Laurer's canal 7 vitellarian duct 8 recepticulum seminis

who worked out the development of *Fasciola hepatica* In 1892 Mont all

others Of recent years our knowledge of the life history of these parasites has been extended in a remarkable manner by Leiper and numerous Japanese observers

**Morphology**—In shape the Trematoda are generally leaf like or tongue shaped and but rarely cylindrical They are provided with a sucker which may have spine-like scales and with one or two suckers oral and ventral

with unicellular salivary glands The intestinal tubules which may be branched



The *Distome cercariae* may be identified as follows —

- A Tails absent—*Cercariae*
- B Tails present —
  - I Tails stumpy—*Monocercous*
  - II Tails well developed —
    - (a) Tails joined forming colony — *Rattenkönig cercariae*
    - (b) Tails not so joined
      - I Tails set with spines—*Trichocercous*
      - II Tails not set with spines —
        - (A) Tails forked at end — *Iurcocercous*
        - (B) Tails not so forked
    - (c) Base of tail forms space into which body can be drawn—*Cystocercous*
    - (D) Base of tail forms no such space —
      - (r) Tail as wide or wider than body—*Rhopalocercous*
      - (r) Tail narrower than body—*Leptocercous*

The *Leptocercous cercariae* may be further differentiated as follows —

- A Body armed anteriorly
  - I With collar and crown of thorns—*Echinostomus*
  - II With a stylet—*Aphidocercariae*
- B Body unarmed anteriorly —*Levinscephalus cercariae*

**Habitat.**—These parasites are found in all classes of the vertebrates and may occur in any of the organs but the most common in man are those of the liver the intestinal tract the lungs and the urinary bladder. It is important to remember that the adult parasites may live in domestic animals especially in cats that they may affect pigs and cattle that development takes place in snails and that the cercariae are free swimming but encystment on grass water weeds etc. must be borne in mind.

be  
an  
the parasite

**Diagnosis.**—The systematic examination by the microscope of the feces after centrifuging especially in cases of diarrhea and of the urine and sputum is the only certain method of diagnosis.

**Treatment.**—The treatment of infections by these animals is little studied but Christopheron has lately recommended Turbithin (vide Chapter LXXXIX and more specially Chapter LXXXII). One may try to kill or expel the intermediate forms by cold reform mixed with eucalyptol or Chenopodium followed by purgation, as described in Chapter LXXX (Ankylostomiasis) or by extract of male fern all in the same chapter (Taeniasis).

**Prophylaxis.**—We do not know enough about the life-cycles to lay down general rules about prophylaxis but it will be obvious

### Watsonius Stiles and Goldberger 1910

**Definition**—Cladorchiine without genital sucker with lobate or lobulate testes without cirrus pouch and with each oral invagination single

**Species**—*Watsonius watsoni* (Conyngham 1904) Stiles and Goldberger 1910

1904)

**Definition**—*Watsonius* with the characters of the genus

**History**—*Watsonius watsoni* was first discovered in the duodenum and upper part of the jejunum of a negro who had come from

Adamawa in late German West Africa to Northern Nigeria. Since its discovery it has been reported near Lake Chad. The type was first described by Conyngham later by Shipley and in 1910 by Stiles and Goldberger.

**Morphology**—The parasite is reddish yellow when fresh 8 to 10 mm.



pair of lateral caudal irregularly globular suctional pouches. The pharynx is spherical with two lateral diverticula called the pharyngeal pouches. The oesophagus divides into two long intestinal caeca about the level of the junction of the anterior third with the posterior two thirds of

FIG. 212.—*Watsonius watsoni*

(Sketch amended by Leiper)

The excretory vesicle is relatively



### Watsonius Stiles and Goldberger, 1910

**Definition**—Cladorchum without genital sucker, with lobate or lobulate testes without cirrus pouch and with each oral invagination single

**Species**.—*Watsonius watsoni* (Conyngham, 1904) Stiles and Goldberger 1910

1904)

**Definition**—*Watsonius* with the characters of the genus

**History**.—*Watsonius watsoni* was first discovered in the duodenum and upper part of the jejunum of a negro who had come from Adamawa in late German West Africa, to Northern Nigeria. Since its discovery it has been reported near Lake Chad. The type was first described by Conyngham later by Shipley, and in 1910 by Stiles and Goldberger.



FIG. 212.—*Watsonius watsoni*  
(After Shipley, amended by Leiper.)

**Morphology**.—The parasite is reddish yellow when fresh 8 to 10 millimetres in length by 4 to 5 millimetres in breadth. In shape it is oval or pyriform. The ventral sucker is large and situated posteriorly and subterminally while the oral sucker is so small as to be hardly worthy of being considered a true sucker. It has a pair of lateral caudal irregularly globular suctional pouches. The

the body and is here surrounded by a sphincter muscle. The excretory pore opens slightly to the left of the middle line dorsal to the posterior sucker. The excretory vesicle is relatively

anterior border where it becomes thick and muscular and is called the 'metatreme'. The eggs are large (122 to 130  $\mu$  in length by 75 to 80  $\mu$  in breadth).

**Life-History.**—The life-cycle is not known but it is believed that

## FAMILY II GASTRODISCIDÆ Stiles and Goldberger, 1910

**Definition.**—Paramphistomoides with rather discoidal bodies divided by a transverse constriction into cephalic and caudal portions. Ventral pouch absent. Venter with many large papillae. Acetabulum ventral at caudal end.

**Type Genus.**—*Gastrodiscus* Leuckart 1877 the other genus is *Homalogaster* Poirier 1883.

### *Gastrodiscus* Leuckart 1877

**Definition.**—*Gastrodiscidae* with bodies slender anteriorly and broadened posteriorly. The latter contains the genital glands. The acetabulum is small. The ventral pouch is absent. The oral sucker has paired evaginations and leads into an oesophagus with muscular thickening. The caeca are long and wavy and end posteriorly. Male organs.—These are two branched testes and a cirrus pouch which is not completely closed. Female organs.—

vitelliferous extracuticular  
is pre-vascular

1876 in the horse

*caudatus* Lewis 1907 in

the horse, *G. minor* Leiper 1913 in the pig in Canada and Nigeria

### *Gastrodiscus hominis* Lewis and McConnell 1876

**Synonym.**—*Amphistomum hominis* Lewis and McConnell

**Definition.**—*Gastrodiscus* 5-8 mm in length and 4 mm broad

**History.**—It was first described in 1876 by Lewis and McConnell  
sucker to the  
ling colon of  
viscera of India

and perhaps it may be common. We have not met with it in Ceylon though there was a small jar in the Medical College Museum labelled *Amphistomum hominis* but without a history.

Leiper has made *G. hominis* the type of a new case of *G. discoidalis* got hold from *Gastrodiscus* by the absence of papillae on the venter and the position of the genital pore on the centre.

**Morphology.**—The parasite is red-flesh-coloured, 8 to 10 millimetres in length and 4 to 5 millimetres in greatest breadth, tapering to 2.5 millimetres in front. The thickness is about 4 millimetres. The posterior end of the body present.



**Pathogenicity.**—It is not known whether these parasites cause any disease. It occurs in 5% of the pigs in French Indo China.

**SUPER-FAMILY FASCIOLOIDEA** Stiles and Goldberger, 1910

**Synonym.**—*Distomata* Retzius, 1782

**Definition.**—Prostomata with acetabulum ventral and always separated from the posterior extremity by some part of the genital apparatus. Oral sucker present, cæca two in number. Hermaphrodites or with separate sexes.

**Type Family.**—*Iasciolidæ* Railliet, 1895

**Classification.**—The super family may be classified into the following families as follows —

**A. Hermaphrodites** —

I. Oral sucker without collar of strong pointed spines —

(a) Ovary in front of testes —

1 Genital pore in front of ventral sucker—*Fasciolidæ*

2 Genital pore not in front of ventral sucker —

(A) Genital pore surrounded by a pseudo sucker—*Heterophyidæ*

(B) Genital pore not so surrounded —

(c) Cuticle with pointed spines—*Troglo-*  
*tremidæ*

(d) Cuticle without pointed spines—*Opsi-*  
*thorchidæ*

(b) Ovary behind testes—*Dicrocoelidæ*

II. Oral sucker with a dorsal and lateral but not ventral fold or collar bearing pointed spines—*Echinostomidæ*

**B. Sexes separate**—*Schistosomidæ*.

**FAMILY FASCIOLIDÆ** RAILLIET, 1895.

**Definition.**—Fascioloidea, hermaphrodites with oral sucker without spiny collar, with acetabulum behind the ventral sucker, cæca two, dorsally and ventrally poorly developed.

**Classification.**—The family is classified as follows —

A. Branches of intestine present, cæca branched —

B. Branches of intestine not present, cæca branched — *Fasciolopsis*

anterior border where it becomes thick and muscular and is called the 'metatremna'. The eggs are large (122 to 130  $\mu$  in length by 75 to 80  $\mu$  in breadth).

**Life-History.**—The life-cycle is not known, but it is believed that the usual host is a herbivorous animal.

**Pathogenicity.**—It may perhaps cause diarrhoea and anæmia. A

Li: . . .  
To u: . . .

## FAMILY II GASTRODISCIDÆ Stiles and Goldberger, 1910

**Definition.**—Paramphistomoidæ with rather discoidal bodies divided by a transverse constriction into cephalic and caudal portions. Ventral pouch absent. Venter with many large papillæ. Acetabulum ventral at caudal end.

**Type Genus.**—*Gastrodiscus* Leuckart 1877 the other genus is *Homalogaster* Poirer 1883.

### *Gastrodiscus* Leuckart 1877

**Definition.**—Gastrodiscidæ with bodies slender anteriorly and broadened posteriorly. The latter contains the genital glands. The acetabulum is small. The ventral pouch is absent. The oral sucker has paired evaginations and leads into an œsophagus with muscular thickening. The cæca are long not wavy and end post-testicularly. Male organs.—These are two branched testes and a cirrus pouch which is not completely closed. Female organs.—Ovary and shell gland are post testicular. Vitellaria are extracæcal,

prevesicular  
876 in the horse  
*undus* Looss 1907 in  
in Uganda and Nigeria

### *Gastrodiscus hominis* Lewis and McConnell 1876

**Synonym.**—*Amphistomum hominis* Lewis and McConnell

**Definition.**—*Gastrodiscus* 5.8 mm in length and 3.4 mm broad

**History.**—It was first described in 1876 by Lewis and McConnell who found it in hundreds attached by its posterior sucker to the mucosa of the cæcum, vermiform appendix and ascending colon of an Assamese. Since then it has been reported in natives of India and perhaps it may be common. We have not met with it in Ceylon though there was a small jar in the Medical College Museum labelled *Amphistomum hominis*, but without a history.

Leiper has made *G. hominis* the type of a new genus *Gastrodiscoides* distinguished from *Gastrodiscus* by the absence of papillæ on the venter and the position of the genital pore on the cone.

**Morphology.**—The parasite is reddish-coloured 8 to 10 millimetres in length and 4 to 5 millimetres in greatest breadth tapering to 2.5 millimetres in front. The thickness is about 4 millimetres. The posterior end of the body presents

There are two suckers—an oral which is situated at the anterior end of the

the  
pen  
vora

The two testes are much branched tubes lying in the middle of the body



FIG 217—THE REDIA OF  
*Fasciola hepatica*

(After Thomas from the *Quarterly Journal of Microscopical Science*)



FIG 218—THE CERCARIA OF  
*Fasciola hepatica*

The branched and tubular ovary lies on the right in front of the anterior testes. The ovarian ducts run backwards to join the middle line with the

the cirrus

... of a ... collar ... and the pharynx from

is along the  
d by a large  
region of the

## SUBORDER FASCIOLINÆ Odhner 1910

**Definition**—Fasciolidae with a shoulder separating the head from the body with a much branched intestine without a receptaculum seminis

**Remarks**—This subfamily contains the genus *Fasciola* which concerns us

*Fasciola* Linnæus 1758

**Definition**—Large Fascioloidea with leaf like bodies with the anterior end shaped into a conical head and with the ventral sucker situated near the mouth. Cuticle covered with spines

**Type Species**—*Fasciola hepatica* Linnæus 1758

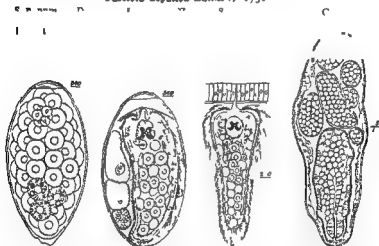
*Fasciola hepatica* Linnæus 1758

FIG 213 — EGG OF *Fasciola hepatica*

FIG 214 — THE MIRACIDIUM OF *Fasciola hepatica*

FIG 215 — THE LARVA OF *Fasciola hepatica*

FIG 216 — THE SPOROCAST OF *Fasciola hepatica*

(After Thomas from the *Quarterly Journal of Microscopical Science*)

**History**—*Fasciola hepatica* the liver fluke is a parasite of sheep, oxen,

**Flukes**

**Morphology**—*Fasciola hepatica* is a flat oval animal with an anterior triangular projection. Length 0 to 30 millimetres, breadth 8 to 13 millimetres. The cuticle covered with minute pointed scales directed backwards.

sides large acetabulum situate close behind oral sucker ]  
15

for

*I hepatic*  
occurred

man for a parasite somewhat resembling it was expelled from lung during a fit of coughing associated with hæmoptysis but there is some doubt as to whether it was not different from Cobbold's species. Its length was 26 to 28 millimetres and its breadth 8 millimetres but it was contracted. It caused fever cough slight hæmoptysis.

#### SUBFAMILY FASCIOLOPSINÆ Odhner 1910

**Definition**—Fasciolidæ without shoulder between head and body with simple zigzag intestines and with a receptaculum seminis.

**Type Genus**—*Fasciolopsis* Looss 1896

#### *Fasciolopsis* Looss 1896

**Definition**.—Fascioline with large ventral sucker elongated posteriorly to form a sac. Cirrus pouch long and cylindrical. Laurer's canal present.

**Type Species**—*Fasciolopsis buski* Lankester 1857

**Classification**—Four species are known to occur in man and they can be recognized as follows—

A *Spines present on cuticle*—

I Vitelline acini very large—*Goddardi*

II Vitelline acini not large—*Huan's fluke* (?)

B *No spines on cuticle*—

I Cirrus sac conspicuous—

(a) Cirrus pouch very long broad convoluted powerfully built—*Julleborni*

(b) Cirrus pouch not so long narrow straight powerfully built—*Buski*

II Cirrus sac inconspicuous—*Rathouisi* (?)

#### *Fasciolopsis buski* Lankester 1857

**Synonyms**—*Distomum buski* Lankester 1857 *D. crassum* Büsch 1859 *nec v.* Siebold 1836 *Distomum rathouisi* Poirier 1887

**History**—*Fasciolopsis buski* is a very large trematode which was first discovered by Busk in the duodenum of a Lascar who died in the Seamen's Hospital in 1843. In 1857 it was named by Lankester and in 1859 described by Cobbold.

It appears to be by no means uncommon in man and pigs in South China and is known in Borneo the Straits Settlements Assam and India. In 1910 *F. rathouisi* Poirier 1887 was regarded

separate because the cirrus sac is convoluted and not conspicu



FIG. 219.—*Limnaea truncatula* MÜLLER.

**Fasciola gigantica** Cobbold, 1856

Synonyms.—*Fasciola angusta* Railliet, 1895, *F. gigantea* Cobbold, 1858

Definition.—Fasciola with short cephalic cone, almost parallel

ovary was branched and it—the shell gland and the yolk glands (which met each other posteriorly)—resembled those of *F. buski*

**Pathogenicity.**—The patient suffered from vomiting which resulted in the expulsion of the flat worms

### *Fasciolopsis goddardi* Ward 1910

**Definition**—*Fasciolopsis* with spines and very large vitellina

**Remarks**—This fluke which is imperfectly known was found in Shanghai China and measures 22 x 9 mm

## FAMILY HETEROPHYIDÆ ODHNER 1914

**Definition**—*Fascioloidea* hermaphroditic with the ovary in front of the testis genital pore behind the ventral sucker and surrounded by a pseudo sucker which is behind or on a level with the acetabulum and has its musculature

classified as follows—

- A Acetabulum and genital suckers ventrally situate and separate—*Heterophyes*
- B Acetabulum and genital suckers dextro laterally situate and surrounded by a complex musculature—*Metagonimus*

### GENUS HETEROPHYES Cobbold 1866

**Synonyms**—*Cotylogonimus* Luhe 1899 *Canogonimus* Looss 1899

**Definition**—*Heterophyidæ* with suckers ventrally situate with a narrow movable anterior portion and a broader less movable posterior portion which contains the genitalia Cuticle with scale like spines suckers widely separated oesophagus long Genital pore placed laterally behind the ventral sucker and surrounded by a genital prominence with chitinous rodlets No cirrus pouch The testes are at the posterior end and the ovary in a median position between them Yolk glands are small and situated at the sides posteriorly

**Type Species**—*Heterophyes heterophyes* v Siebold 1852

### *Heterophyes heterophyes* v Siebold 1852

**Synonyms**—*Distomum heterophyes* v Siebold 1852 *Mesogonimus heterophyes* Raillet 1890 *Cænogonimus heterophyes* Looss 1900 *Cotylogonimus heterophyes* Braun 1901

**Definition**—*Heterophyes* with ventrally situate and separate acetabular and genital suckers

common. Lempert has recorded several cases in Chinese seamen

intestinal caeca are not branched and extend to the posterior border. The genital pore is situated anteriorly to the acetabulum but the most remarkable feature is the very long cirrus about one-fourth the length of the body. The testes lie posteriorly with the ovary and the uterus in front. The yolk granules are extensive like those in *F. hepatica*.

**Life History**—The eggs are 0.12 to 0.13 millimetre in length and 0.077 to 0.08 in breadth and the larval stages are said to occur in shrimps.

**Habitat**.—The intestine of the pig and man.

**Pathogenicity**—It is believed to cause dysenteric diarrhoea, wasting and jaundice at times.

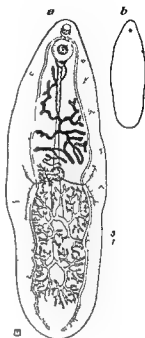


FIG. 210.—*Fasciolopsis buski*.

**Fasciolopsis fullerborni** Rodenwaldt  
1909

**History**—This worm was discovered and described by Rodenwaldt in 1909, being found in the motions of an Indian in Hamburg.

**Morphology**—The parasite is very large, measuring from 30 to 50 millimetres in length and from 14 to 16 millimetres in breadth. The oral sucker and acetabulum are with

The eggs developed. The eggs measure 0.1 millimetre in length by 0.073 millimetre in breadth.

**Habitat**.—Intestine of man.

**Pathogenicity**—The patient harbouring it was suffering from fever which had been diagnosed as typhoid.

**Kwan's Fluke** (*F. goddardi*?)

Under the name of

It was

the posterior end.  
The



diameter. The testes are elliptical and situate posteriorly. The circulatory ducts open with the uterus into a genital sinus which opens into a pit at the front of the ventral sucker.

The ovary is spherical and lies in the middle of the posterior part of the body. A receptaculum seminis and a Laurer's canal are present. The vitelline acini lie on each side in the posterior part of the body. The uterus forms three to four transverse coils.

**Life-History**—The eggs are elliptical  $28 \times 16$  microns, double contoured and yellowish brown with an operculum but no shoulder and a knob at the narrower end.

Muto found cercariae in the liver of *Melania libertina* in Kaishu. The cercariae were found in the liver under the skin and fed with

The cercariae also live in a trout *Plecoglossus altivelis* rarely in other fish. Infection takes place by eating raw fish and the period judging by dogs is seven to sixteen days for the eggs to appear in the faeces.

**Pathogenicity**.—It causes chronic diarrhoea in man.

#### FAMILY TROGLOTREMIDÆ ODHNER, 1914

**Definition**—Tascioloidea hermaphroditic with ovary in front of testes, genital pore just in front or just behind rim of acetabulum but not surrounded by a pseudo-sucker and with the cuticle completely covered with pointed spines. Vitellaria well developed and for the most part dorsally situate. More or less flattened worms 2-3 mm in length with extreme posterior end prolonged into a small appendage with ventral surface flat or somewhat hollowed and dorsal surface vaulted. Musculature in forms living in cysts poorly developed. Pharynx present. Gut diverticula present. Genital pore of the acetabulum usually

absent. Pars prostatica and seminal vesicle present. Testes symmetrical. Ovary in front of the testes. Vitellaria well developed. Uterus long and much coiled with small eggs or short an

in *Zoologiska Bidrag*  
the frontal sinuses of  
ts in the pylorus of

*Phocæna communis* for *Collyricium java* and *Sylva hortensis* and for *Paragonimus ringeri* etc.

**Type Species**—*Troglorema* Odhner 1914

#### GENUS PARACONIMUS BRAUN 1899

**Definition**—Trogloremidæ with thick oval or broad fusiform bodies almost circular on transverse section. Cuticle with sac-like spines. Suckers separated by half the length of the body.

It is also found in dogs and cats in Egypt Japan and Formosa in man in Khartoum (eggs = 0.26 x 0.013 mm) also adults and in dogs in the same town

**Morphology**—*Heterophyes heterophyes* is pear shaped very narrow in



FIG. 21.—*Heterophyes heterophyes*

(After Looss from Braun's Animal Parasites of Man, English edition)

a Schematic  $\times 15$  b natural size c eggs  $\times 250$  d spine  $\times 700$

be overlooked

GENUS METAGONIMUS Katsurada 1913  
SYNONYM  $\times 2$

suckers

Katsurada

*Metagonimus yokogawai* Katsurada 1913

Synonym—*Yokogawa yokogawai* Leiper 1913  
Leiper now believes that this fluke is the same as one previously described by Kobayashi as *Ovotrematoides ovatum* n. g. n. sp.

Definition  $\times 2$

with body length with a the body and

the part of the they enter and

like possessing 25 microns in

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The intestinal cæca are wavy and run to the posterior end of the body but are unbranched. The excretory vesicle runs from the pharynx backwards to its aperture on the posterior margin of the body. The genital pore is just behind the ventral sucker. There is no cirrus pouch and no receptaculum seminis.

fr.

icl.

three may be distinguished as follows but Japanese workers do not accept these characters as of specific value —

A Chisel shaped spines —

I Arranged in circular rows in groups—*Ringeri*

II Arranged in circular rows singly *Kallicotti*

B Lancet shaped spines —

Arranged in circular rows in groups—*Westermanni*

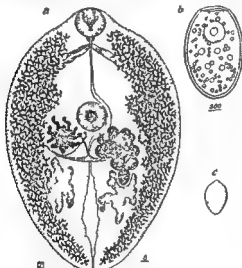


FIG. 22.—*Paragonimus ringeri*

(After Leoss from Mense's Tropenkrankheiten.)

a Schematic  $\times 5$  b eggs  $\times 300$  c natural size

### *Paragonimus ringeri* Cobbold 1880

**Synonyms**—*Distoma ringeri* Cobbold 1880 *D. hepaticum* Miura  
*D. pulmonale* Baelz 1883 *D. pulmonis* Suga 1883 *D. cerebralis*  
1889 Yamagiwa 1890 *Mesogonimus westermanni* Railliet 1891  
*M. pulmonis* Stossich 1892

**History**—*Paragonimus westermanni* was discovered by Herbert in 1878 in the lungs of two Pungal tigers which died respectively in the Zoological Gardens of Amsterdam and Hamburg.

posterior lobate. The ovary is slightly lobate and it and the uterus situated medianly. The yolk glands lie laterally in the middle third, extending from behind the ventral sucker to the back of the testes. There is a cirrus pouch. The eggs are oval (0.034 by 0.021 millimetre).

Habitat—The dog and man

Pathogenicity—Not known

### Clonorchis Looss 1907

**Definition**—Opisthorchunæ characterized by the fact that the testes are not notched or lobate but distinctly ramified, the branch crossing the intestinal caeca on their ventral side and extending very near the body margin. The excretory vesicle is simply an unpaired tube which becomes somewhat widened at its anterior end, assuming sometimes the shape of an irregular triangle.



FIG. 223—*Clonorchis sinensis*

(After Looss from the *Annals of Tropical Medicine and Parasitology*)

a Schematic b natural size c egg  
X 500

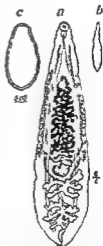


FIG. 224—*Clonorchis endemicus*

(After Looss from the *Annals of Tropical Medicine and Parasitology*)

a Schematic b natural size c egg  
X 500

**Species**—*Clonorchis sinensis* Cobbold 1875 *Clonorchis endemicus* Baelz 1883 but the work of Kobayashi in 1917 tends to show that they are one and the same species

### *Clonorchis sinensis* Cobbold 1875

**Synonyms**—*Distoma sinense* Cobbold 1875 *D. spathulatum* Leuckart 1876 *Distomum hepatis innocuum* Baelz 1883

**Definition**—*Clonorchis* with the generic characters.

***Opisthorchis viverrini* Poirier 1886**

**Definition**—*Opisthorchis* with cuticle covered with minute acicular spines. Ovary multilocular. Branches of intestine almost reach the hinder end of the body and the ovary and testes are deeply lobed.

**Remarks**—This trematode which belongs to the Indian civet cat was obtained by Kerr from prisoners at Chiengmai and recognized by Leiper.

**Infection**—Infection is probably by eating raw or partially cooked fresh water fish.

***Paropisthorchis* Stephens 1912**

**Definition**—*Opisthorchinæ* with lobed testes and with ventral process on which are situate the ventral sucker and the genital pore.

**Type Species**.—*Protropisthorchis caninus* Barker 1912

***Paropisthorchis caninus* Barker 1912**

**Definition**—*Paropisthorchis* with the generic characters.

**Remarks**—Cobbold in 1858 found a little fluke *Distomum conjunctum* in the bile ducts of *Canis fulvus* Lewis, the American fox.

Fourteen years later Lewis and Cunningham found the same fluke in Indian pariah dogs and in 1874 McConnell found what was thought to be the same fluke in human beings in Calcutta. In 1903 Braun pointed out that the American and Indian flukes were different and named the latter *Opisthorchis noveboracensis* (*vide infra* Amphimerus).

In 1912 Barker separated the parasite of the Indian pariah dog from the human calling the former *O. caninus* and in the same year Stephens created a separate genus for it. Leiper still maintains however that these two forms in man and dog are identical.

It is not known to occur in man.

***Amphimerus* Barker 1912**

**Definition**—*Opisthorchinæ* without ventral process but with lobed testes and vitellaria divided by the position of the ovary into anterior and posterior lobes.

**Type Species**—*Amphimerus noveboracensis* Braun 1903.

**Remarks**—This genus was created for Braun's *Opisthorchis noveboracensis* which as explained above according to Stephens only applies to McConnell's flukes found in two Mohammedans in Calcutta.

***Amphimerus noveboracensis* (Braun 1903)**

**Definition**—*Amphimerus* with the characters of the genus.

**Morphology**—

pointed body ca.  
2.5 millimetres in  
being larger than  
ventral sucker  
far back. The t

mediate host The first intermediate host and the method of infection of the fish are unknown but *Melanis libertina* is suspected

Habitat—It is found in cats dogs hogs and men

Pathogenicity—Enlargement of the liver and diarrhoea

#### A Possible Feline Clonorchis (or Opisthorchis)

Looss draws attention to the fact that Ijma in 1886 describes a *Distomum*



FIG 225—*Dicrocoelium dendriticum* Rudolphi

(After Looss from Mense's Tropickrankheiten)

a Schematic  $\times 6$  b natural size c eggs  $\times 250$

he mentioned three found in a man from the province of Saga with an average length of 5.16 millimetres and breadth of 0.96 millimetre which Looss considers can only be explained as an infection with a feline species

Habitat—Cats and man (?)

### FAMILY DICROCELIIDÆ

OHNER 1910

Definition—Fascioloidea hermaphroditic without spiny collar around the oral sucker and with the ovary behind the testes

Type Genus—*Dicrocoelium* Dujardin 1845

*Dicrocoelium* Dujardin 1845

Definition—*Dicrocoelidæ* with lanceolate shaped bodies without spines and with suckers placed close together. Intestine does not reach the posterior end. Genital pore close behind the pharynx with a cirrus pouch in front of the ventral sucker just behind which the testes lie with the ovary in the median line behind them. The uterus lies behind

the ovary and testes extending as far back as the posterior border. The yolk glands are small and situated in the middle quarter of the lateral areas of the body. The excretory vesicle is tubular. Ova dark brown. Worms live in the liver and gall bladder rarely in the intestine

Type Species—*Dicrocoelium dendriticum* Rudolphi 1819

*Dicrocoelium dendriticum* Rudolphi 1819

Synonyms—*Fasciola lanceolata* Rudolphi 1803 nec Schrank 1790 *Distomum lanceolatum* Mehlis 1825 *Dicrocoelium lanceolatum* Dujardin 1845 *D. roscali lanceolatum* Stiles and Hassall 1896

**History**—*Clonorchis sinensis* was first discovered by McConnell in 1874 in the liver of a Chinaman. It was believed to be an *Opisthorchis* but in 1907 Looss gave reasons why it should be placed in the new genus *Clonorchis*. As far as is known it occurs principally in China and Japan and has not yet been found in animals.

**Remarks**—It may be the same as *Clonorchis endemicus* Baelz 1883 *Distoma hepatis endemicum sive perniciosum* Baelz 1883 *Distoma japonicum* R. Blanchard 1886. In 1883 flukes were first described in the liver of human beings in Japan by Kiyono Nakahama Suga and Yamagata and a little later in the same year Baelz reported the occurrence of two hepatic distoma in Japan.

1 inch h

“ “

differences

It is found in Japan where it is common and in Annam and Tonkin in man cats dogs and pigs

“

h d h own h

breadth

the infected flesh. He also found cysts in other fish—*Clonorchis lanceolatus* *A. limbatus* *A. cyanodigmus* *Paracheilognathus rhombus* *Pseudoperclampus typus* *Abbottina psegma* *Breia zezera* and *Sarcocheilichthys variegatus*. These fish are the second inter



of the acetabulum but not beyond it while that of *st. stratiotes* reaches 0.75 mm. therefore it is not a member of the Echinostominae

#### GENUS ECHINOSTOMA Rudolphi 1809

**Synonym** — *Fasciolella* Garrison 1908

**Definition** — Echinostominae with small elongated bodies broader anteriorly than posteriorly with characteristic circumoral ring of spines and with other spines on the body and with large prominent acetabulum. Oesophagus short intestinal caeca unbranched. Excretory vesicle tubular. Genital pores anterior to the acetabulum. Testes compact situate in the median line one behind the other. Cirrus and pouch well developed. Ovary compact no receptaculum seminis. Laurer's canal present. Yolk glands well developed in the posterior fifth of the body well-developed shell gland and uterus which lies between the ovary behind and the acetabulum in front. Ova large and operculated.

**Remarks** — With regard to the position of *Echinostoma* it should be observed that some authorities do not classify it under the Fasciolidae but under the Echinostomidae which we adopt. The species *Echinostoma revolutum* (Froese) is *Distoma eclanati* n. Zeder 1803. Another species of importance in tropical medicine is *F.ilocanum* (Garrison 1908).

#### *Echinostoma ilocanum* Garrison 1908

**Synonym** — *Fasciolella ilocana* Garrison 1908

**History** — *Echinostoma ilocana* was discovered and described by Garrison who in 1907 noticed peculiar eggs in the feces of Philippine prisoners in Bilbid Prison in Manila and subsequently after treatment with male-fern obtained a small number of trematodes. Quite recently Odhner has shown that it belongs to the genus *Echinostoma* family Echinostomidae.

to the  
The  
fairly

**Life-History** — Nothing is known of the life history beyond the fact that a miracidium hatches in about ten days after the eggs have left the host.

**Habitat** — The intestine of man in Luzon in the Philippine Islands.

**Pathogenicity** — It is probable that the worm is non pathogenic.

**History**—Bucholz appears to have been the first to discover these worms in the gall bladder in Weimar and later Chabert in r Since found in Europe

**Morphology**—*Dicrocoelium dendriticum* is a small trematode measuring 8 to 10 millimetres in length by 1.5 to 2.5 millimetres in breadth. It is pointed in front and narrow behind so that the widest point is just behind the yolk glands. Cuticle is smooth, the oral sucker is terminal and about the same size as the ventral (0.5 to 0.6 millimetre). The intestine bifurcates just in front of the genital pore which is situated in the median line in front of the ventral sucker behind which the two testes lie from which the vasa deferentia

**Life-History**—Not known but suspicion rests on *Planorbis marginalis* and land snails

**Pathogenicity**—No special symptoms

#### FAMILY ECHINOSTOMIDÆ LOOSS 190

**Definition**—Fascioloidæ hermaphroditic with a fold or collar bearing a row or rows of pointed spines on the dorsal and lateral ally for

as follows —

- A. Cirrus sac does not reach beyond acetabulum. Without strong rosethorn hooks—*Echinostominae*
- B. Cirrus sac reaches beyond acetabulum. With strong rosethorn hooks—*Himasthinae*

#### SUBFAMILY ECHINOSTOMINÆ LOOSS 1899

**Definition**—As above

**Type Genus**—*Echinostoma* Rudolphi 1809

**Classification**—*E. malayanum* Leiper 1911 has been placed by Odhner in the genus *Euparyphium* Odhner so that it now becomes *Euparyphium malayanum* as according to Leiper it is probably the same

fc  
A  
pc  
Lane pointed out that as Odhner's principal character of the Echinostominae was that the cirrus sac usually reaches to the centre



## GENUS EUPARYPHIUM Odhner

*Euparyphium malayanum* Leiper 1911

Synonym — *Echinostoma malayanum* Leiper 1911

History — In 1911 Leiper received two consignments of flukes from Dr Macaulay of Singapore and from Dr Stanton of Kuala Lumpur which had been collected from the intestine of a Tamil in the Malay States

March 1911

the body

The door

and large in size

Habitat. — Leiper does not think that this worm is a normal parasite of man and considers that domesticated animals especially cats and dogs should be investigated for it

Pathogenicity — Not stated

## SUBFAMILY HIMASTHINÆ Odhner 1910

Definition — Echinostomidae in which the cirrus sac extends caudad to the acetabulum

Remarks — The only genus with which we are concerned is —

## GENUS ARTYFECHINOSTOMUM Clayton Lane 1915

Definition. — Himasthinae without strong rosethorn hooks

Remarks — There has been much dispute as to whether this is a good genus or not

Type Species — *Artyfechinostomum sufrartylax* Clayton Lane 1915

*Artyfechinostomum sufrartylax* Clayton Lane 1915

Nomenclature — Leiper has pointed out that this name clashes with the recommendations of the International Rules. The use of proper names in the formation of compound generic names is objectionable. However the name cannot now be changed

History — The worm was found in a girl aged eight years on the Ragnik Tea Estate in Assam and was thought by Leiper to be probably the same as *Euparyphium malayanum* but in 1917 Clayton Lane showed that the cirrus sac extended 0.75 mm caudad to the acetabulum a fact agreeing with the definition of the subfamily

to the gills and the wall of the alimentary canal where in twelve days the first rediæ appeared and gradually concentrated in the bile ducts where they grew and formed second rediæ. Mice were immersed in the water in which the full grown snails were kept and after three weeks many *S japonicum* specimens were found in their livers.



FIG. 237.—EGG OF *Schistosoma japonicum* (After Leiper)

In 1914 Leiper confirmed this work in Japan having recognized morphologically the cercaria originating in sporocysts misnamed rediæ by the Japanese authorities. The mollusc in question was named *Katayama nosophora* *Katayama* a synonym of *Blanfordia*.

In 1916 Narabayashi showed that man, cattle, horses, goats, pigs, cats and dogs are naturally infected by *S japonicum* which contains ferments which can digest albumen in an alkaline medium. He also found that in dogs and guinea pigs after penetrating the skin most of the parasites enter the venous blood stream though some few enter arteries or lymphatics. Eventually they all enter the right side of the heart and are scattered all over the lungs at the bases of which they collect and then migrate through the mediastinum, diaphragm and liver into the portal system while a few re-enter the circulation and reach the rectum.

**Morphology.**—In general it resembles *S. haematobium* but the cuticle of the female is smooth and that of the male has only the few spines mentioned above. There are no bosses.

*Male*—8 r . . . . .

which opens just behind the acetabulum. Seminal vessels present.

*Female*—8.26 millimetres in length and about 0.4 millimetre in breadth. Suckers armed with fine spines. Body thicker behind ovary behind which gut forks unite. Ovary elliptical dilated behind where the oviduct arises and running forwards joins the vitellarian duct. Vitellaria well developed lying behind the ovary, but not quite reaching the posterior end of the body.

duct opens

e soft faintly small ip-like

thickenings

The miracidia hatch out quickly and develop into cercariæ in

1864 in a patient from the Cape of Good Hope. Since then it has been found widely distributed through Africa (Egypt Sudan Tunis Algiers West Coast including the Gold Coast East Coast and South and Central Africa) which is its endemic centre and in Asia in Syria Arabia Mauritius Persia India. Only imported cases occur in Ceylon.

Other species are known in sheep horses oxen etc.

In 1915 Leiper discovered its complete life history in the snail and its method of entry into the vertebrate.

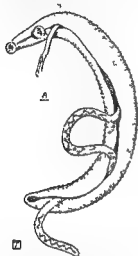


FIG. 26.—*Schistosoma hamatobium*  
(After Loos from Mense's Tropenkrankheiten)

This drawing shows the female worm enclosed in the gynæcophoric canal of the male.

**Morphology.—Male**—The male is whitish in colour and from 12 to 14 millimetre in length with a greatest width behind the ventral sucker of 3 millimetre. It is really thin and flat though it may look cylindrical because the lateral margins are turned ventrally inwards enclosing a canal called the gynæcophoric canal in which the female lies. The whole body is covered



FIG. 27. EGG OF *Schistosoma hamatobium* (250)  
(After Loos from Mense's Tropenkrankheiten)

with projections tipped with short spines which pierce into the wall of all of the bloodvessels. The oral sucker looks ventrally. The ventral sucker is situated near to it. There is no pharynx but the oesophagus is long and

is situated behind the ventral sucker at the beginning of the gynæcophoric canal.

Female

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of t  
anc  
mi

— hamatobium



*Blanfordia nosophora* The cercaria is barrel shaped tapering towards the anterior end mouth with two short lancet shaped bristles Small ventral sucker at posterior one-sixth of body Hinder end with three pairs of poison glands from each of which a duct runs forwards to open into the mouth Two pairs of laterally placed flame cells with vessels In the middle of this body there is an oval light brown body with a small anterior canal The cercariae penetrate the skin pass via the veins to the heart and so to the lungs from the bases of which they penetrate the mediastinum diaphragm liver and so enter the portal vein from which the eggs pass to the submucosa and mucosa of the colon and cause growths The adult worms can live at least two years in the vertebrate

**Pathogenicity**—The cercariae while entering the skin cause the disease *habure* and in the body *katayama disease*

#### Christophers and Stephens' Schistosoma

Christophers and Stephens in 1905 described a *Schistosoma* egg which  
 new and as yet  
*S. japonicum*  
 was found in  
 long snout like

#### New Schistosoma

In 1904 Salomone and Bell found portions of a worm which they think may be a new *Schistosoma* in a patient suffering from hæmaturia contracted in Brazil It may however have been a *S. matroni* which very occasionally occur in the bladder wall and is a common infection in Brazil

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 No 49 II 1914 No 86



## CHAPTER XXV

# CESTOIDEA

Cestoidea—History—Morphology—Life-History—Habitat—Classification—  
Cestodes in man—References

### CESTOIDEA RUDOLPHI, 1808

**Definition.**—Platyhelms without alimentary canal in any stage of the life-cycle with segmented body, in which the epidermis which has sunk into the parenchyma secretes a thick cuticle. Lime-secreting cells are developed in greater or less number and form calcareous cor- and that of th  
that of another host

**History.**—It is believed that cestodes were known to the ancients and that the reason why Moses who figures largely in the history of Tropical Medicine forbade the Israelites to eat pigs and such animals was because of the parasites known to exist in their flesh.

Aristotle knew the proglottides of tapeworms and as early as 1592 *Tænia* was distinguished from *Bothriocephalus*. Tyson (1682) discovered the head of the tapeworm of a dog. Redi (1687 1702) came to the conclusion that *Cysticercus* were animals and Zeder (1800) formed them into a separate group, *Cystica* but Kuchenmeister in 1851 proved by feeding experiments that these were only the larvæ of tapeworms and that as a rule two different kinds of animals were required as hosts in order that the life cycle might take place.

Leuckart, Braun Fuhrmann Looss Sansino Grassi Blanchard von Linstow, Luhe Stiles Leiper and Sambon may be mentioned as investigators who have greatly improved our knowledge of these parasites.

and the Cestodans

becoming narrower and narrower in the middle is readily while

*Opisthorchis noverca*

- BARKER F D (1911) Archives de Parasitologie XIV 13 61 Paris  
(Deals with the whole genus)  
COBBOLD (1859) Journal of the Linnæan Society of London Zool vol V  
p 8

*Paragonimus westermani*

- BAELZ (1880) C f Med Wiss p 721 Berlin  
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krankheiten Orig nale LXI 389 Jena  
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SAMBOY (1908) Journal of Tropical Medicine p 29 (1909) No I p 1  
DA SILVA (1909) Archives de Parasitologie VIII 2 p 281

*Schistosoma japonicum*

- CATTO J (1905) "  
KATSURADA (190  
LEIFER AND ATE  
LOOSS (1905) C  
WOOLLEY (1906)

which the uterus becomes saturated with eggs, the ovum and become branched and grows, filling up the proglottis while the male generative organs atrophy and disappear (vide Fig 253 p 614)

**Life-History**—The proglottis can fertilize itself with or without the use of the cirrus or different proglottides may fertilize one another. In any case the receptaculum seminis receives the spermatozoa which travel down the spermatic duct and meeting the

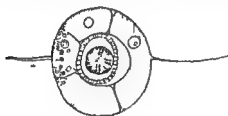


FIG. 240—UTERINE EGG OF *Taenia saginata* (X 375)  
(After Leuckart)



FIG. 241—OVUM OF *Dibothriocephalus latus*  
(After Leuckart)



FIG. 242—FÆCAL EGG OF *Taenia solium* (X 300)  
(After Leuckart)

ovum fertilize it. The fertilized ovum now obtains its yolk (vitellus) and its shell and then passes into the uterus from which it may escape by the uterine orifice when there is one or not until the proglottis is destroyed.

Development usually begins in the uterus. An egg is as a rule oval in form enclosed in a brown or yellow shell with or without an operculum. This shell contains food yolk and the developing embryo whose cells form two membranes—an outer in contact with the shell and an inner in contact with the embryo.

... which keeps it attached to the



protective

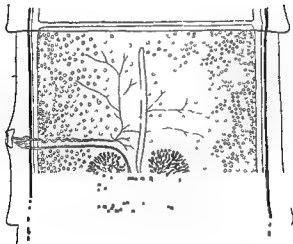


FIG 239—MATURE SEGMENT OF *Tænia siginata* GOEZE 1782  
(After Lenckart)

In the cortex come the longitudinal muscles beneath which are the trans-

like *Hymenolepis murina* which is said to have its larvæ in the villi and its adults in the intestine of the rat infest only one host

Often the *Cysticercus* is found in a herbivorous animal while the

an adult tapeworm  
but may be much  
*fasciolaris* of the mc

The *Cestoidea* Rudolphi 1809 are divided into *Cestoidaria* Monticelli

segments The subclass Cestoda are divided into orders as follows —

#### Cestoda Sensû Stricto Monticelli 1897

**Synonyms** — *Pollaplassogones* E Blanchard *Cestodes digeneses* van Beneden *C. polyzoa* Lang

**Definition** — Cestodes in which the adult worm consists of a scolex and proglottides

**ORDER I PSEUDOPHYLLIDEA** Carus 1863 — **Synonyms** — *Bothriocephaloidea* — Scolex armed or unarmed with two usually slightly developed groove like suckers with three genital orifices Vitellaria situated laterally Eggs with or without a lid

**ORDER II TETRAPHYLLIDEA** Carus — Scolex armed or unarmed with four very motile pedunculated or sessile bothridia or with four round suckers no uterine orifice cirrus and vagina open at the sides Vitellaria situated laterally

**ORDER III CYCLOPHYLLIDEA** 1811 *Beneden* — Scolex with four or five apical rostellum with or without uterine orifice vitellogene gland Eggs without lids  
s — Head stalk armed with hook  
o bothridia

Scolex with two or four

found in man are I

and III

The outer envelope and the shell are soon lost, and when the embryo appears in the faeces it is surrounded by its inner envelope,

which is not an egg at all but the onchosphere with its embryonic envelope which in certain species may be ciliated

When the onchosphere enters the alimentary canal of a new host generally of a different class from the original host it

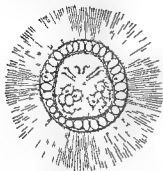


FIG 243—FREE CILIATED EMBRYO  
of *Dibothriocephalus latus*  
(After Leuckart)



FIG 244—CYSTICERCUS OF *Tanis saginata* GOEZE 1782  
(After Leuckart)

throws off its envelope and enters the tissues by its



FIG 245—PLEROCERCOID OF *Dibothriocephalus latus*  
(After Leuckart)

may produce unpleasant symptoms if there are a number of parasites. The time occupied in the transformation of an onchosphere into a *Cysticercus* varies being from two to six months or longer.

With but rare exceptions the *Cysticercus* does not develop further until it enters another and different host, though some,

Sparganum Diesing 1850

A  
have  
can  
*S. baxters*

which  
they  
reproduce

*Sparganum mansoni* Cobbold, 1883

Synonyms.—*Ligula mansoni* Cobbold 1883, *Bothriocephalus liguloides* R Leuckart 1886, *Dibothrium mansoni* Anola, 1900

a  
an  
cavity Scheube discovered another specimen in the urethra of a Japanese and Sonsino a third in an Egyptian jackal

Morphology.—Long white ribbon shaped parasites with feeble movements No head or definite structure visible Length 30 to 35 centimetres, breadth, 3 to 12 millimetres At the broader end there is a sort of papilla There is no reproduction by fission

*Sparganum proliifer* Ijuma, 1905

Synonym.—*Plerocercus proliifer* Ijuma 1905

In 1905 Ijuma found this worm in a woman living near Tokio and in 1907 Gates found the same or a similar worm in a man in Manatee U S A

Morphology.—The larva which lies enclosed in a cyst, may attain 1 to 12 millimetres in length by 2.5 millimetres in breadth The head at the narrow end is motile and capable of evagination and invagination It shows an apical depression, which perhaps serves as a sucker, but there are no true suckers



FIG 249 — *Sparganum proliifer* IJUMA (After Stiles)

muscular and excretory system and reserve bodies It can multiply by transverse division and supernumerary heads may become independent and may assume bizarre and irregular shapes The adult is unknown

Habitat.—It lives in the subcutaneous tissue and elsewhere in man

Pathogenicity.—It produces nodules in the skin and fascia beneath

which may lead to the escape of a worm from the spot When a nodule is cut open a cyst is found containing one or two worms either filled with a watery fluid or with a jelly or slime like sub-

CESTODES IN MAN

The cestodes which are found in man may be classified as follows —

Order and Family	Subfamily	Genus and Subgenus	Species			
Pseudophyllidea	Dibothriocephalidae	Dibothriocephalus	1 D latus			
			Diplogonoporus	2 D cordatus		
				3 D parvus		
		Ligulidae	Sparganium	4 D grandis		
				5 D brauni		
				6 S mansoni		
				7 S prolifer		
				8 S baxteri		
		Cyclophyllidea	Tæniidae	Dipylidinae	9 B jassynensis	
					Hymenolepis	10 D caninum
Drepanidotaenia	11 H nana					
	Davaineinae				Davainea	12 H diminuta
Tænia				13 H lanceolata (?)		
				Tæniarhynchus		14 D madagascariensis
Echinococcus						Tænia
				Echinococcus		
Echinococcus						Echinococcus
	Echinococcus			Echinococcus	18 T atricana	
Echinococcus		Echinococcus	19 T hominis			
	Echinococcus		Echinococcus	20 T philippina		
Echinococcus		Echinococcus		21 T confusa		
	Echinococcus		Echinococcus	22 T bremneri		
Echinococcus		Echinococcus		23 E granulatus		
	Echinococcus		Echinococcus	24 E multilocularis		

Two tapeworms are not included in this list—*Tænia* (*Tænia*) *teniaformis* Bloch 1750 a tapeworm found in cats and *Tænia* (*Tænia*) *pisiformis* Bloch 1780 a tapeworm found in rabbits—because no definite evidence exists that man has ever been infected by them

At the time of —

*nana* (?) *Davainea madagascariensis* *D asiatica* Other hosts may yet be found

*ceph*  
(tri  
the rat and mouse) *Drepanidotaenia lanceolata* (true host ducks and geese)

Therefore in man there is the question of intestinal tæniases to be considered. But cysts may also develop in man from *T solium* and *Echinococcus granulatus* and *multilocularis*. From the species of *Sparganium* larval forms give rise to somatic tæniases. These affections with their treatment will be considered later as will the treatment

It must be not as a — as



Uterus becomes divided into ovarium follicles or entirely atrophied and the eggs are set free into the parenchyma. Eggs with thin transparent shells with or without appendages.

Genera—(1) *Dipylidium* (2) *Hymenolepis*

### *Dipylidium* Leuckart 1863

Definition—*Dipylidium*, of medium or small size with retractile rostellum armed with several rings of alternating hooks which have a broad basis. Gentrals duplicated with pores on each side of a proglottis. Eggs with a double shell.

### *Dipylidium caninum* Linnæus 1758

Synonyms—*Tænia canina* Leuckart 1758 *Moniliformis* Pallas, 1781 *T. cucumerina* Blochmann 1782 *T. elliptica* Batsch 1786

having been first found by Dubois a pupil of Linnæus in 1751. Melnikow showed that the scolex was to be found in the dog louse (*Trichodectes canis*) and in that of the cat (*T. subrostratus*) as well as in the dog flea (*Ctenocephalus canis* Curtis) the cat flea (*C. felinus* Bouche) and in that of man (*P. irritans*) but how the infection

reaches man is not definitely proved. Recently Blanchard has summarized sixty cases in man up to the year 1907 most of which occur among young children.

**Morphology**—It measures 15 to 35 and even to 40 centimetres in length and the segments are from 1.5 to 3 millimetres in breadth.

The scolex has the typical rostellum with three to four rings of hooks diminishing in size from the first circle where they are 12 to 15  $\mu$  to the last



FIG. 251.—*Dipylidium caninum*: nest of eggs ( $\times 250$ )

(From a photograph by J. J. Bell)

where they are only 5 to 6  $\mu$  in length and four suckers which are unarmed.

each side. The eggs are unshelled.

on their own movement pass through the intestines where the eggs infect and develop into cysts.

on the cysticercus to 1 a very small size gets infected in that manner.

itself and may pass through the cat licks its fur and

stance Apparently after weeks or months the cyst wall may become firm and thick and encapsulate the worms This condition may last for years and apparently can kill the victim who may be literally eaten up of worms

**Sparganum baxteri Sambon 1907**

This Dibothriocephalid larva was found by Baxter in an abscess in the thigh of a Masai in late German East Africa



FIG 250—*Sparganum baxteri* SAMBON  
(After Sambon)

Morphology  
length  
verse 1  
face Anterior extremity is 2 millimetres broad with a completely invaginated head posterior extremity 1.5 millimetres broad with a shallow medium slit

Leon has described a new tapeworm in man which is referred to the Ligulinae in which it requires a new genus called *Bransia jussyei* Leon 1908

**ORDER III CYCLOPHYLLIDEA van Beneden**

There is only one family with human parasites

**FAMILY TENUIDÆ Ludwig 1886**

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St  
Tenuidæ

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maly 3

**SUBFAMILY I DIPYLIDIINÆ Stiles 1896**

Definition—Tenuidæ with armed rostellum and unarmed suckers Genital pores marginal Genitalia simple or double

**Hymenolepis (Hymenolepis) nana** von Siebold 1852

Synonyms—*Tænia murina* Dujardin 1845 *nec* Gmelin 1790  
*T. nana* von Siebold 1852 *nec* van Beneden 1867 *T. ægyptiaca*  
 Bilharz 1852 *Diplacanthus nanus* Weinland 1858

This is the dwarf tapeworm of man and was discovered by Bilharz in the ileum of a boy who died of meningitis in Cairo. It is found in Egypt, Europe, North and South America, Siam and Japan and is by no means a rare parasite in man. Calandruccio estimates that 10 per cent of the children in Sicily are affected. Stiles reports 4 per cent for 125 children in Washington. The number found varies from one or two to several thousands in one individual.

There is doubt as to whether this worm is or is not identical with *Hymenolepis nana* var. *fraterna* Stiles 1906 which is common in rats and mice. Probably it is distinct from this parasite because Grassi and Looss were unable to transmit it to mice.

**Morphology**—It is the smallest tapeworm in man measuring only 10 to 15 millimetres in length and from 0.5 to 0.7 millimetre in breadth.

The head is globular and provided with a rostellum with one ring of twenty-four to thirty hooks which are very small, 14 to 18  $\mu$  in length.

The mature uterus contains about thirty or more eggs which are oval and measure 30 to 48  $\mu$  and contain onchospheres measuring 16 to 19  $\mu$  in diameter.

**Life-History**—It is believed that the cysticercus will be found in some insect.

The nearly related *T. nana* var. *fraterna* completes its development entirely in the rat for the cysticercus develops in the villus while

poorer families. Apparently when the parasite occurs in small numbers no symptoms result but in considerable numbers disturbance may occur which has been assigned by Mingazzini to the effects of toxins.

**Treatment**—Male fern is the best treatment (*vide* Chapter LXXV).

insisted upon

**Subgenus 2 Dropanidotænia** Railliet 1892

With the characters of the genus but the hooks have a dorsal root much larger than the ventral and are only eight to twenty in number. Proglottis broad, testes three alongside which lies female genitalia.

Blanchard thinks that the cat spreads the parasite by infecting milk with the cysticercoids when it steals a drink from a bowl the contents of which are afterwards given to children

### Hymenolepis Weinland 1858

Synonym.—*Diplacanthus* Weinland 1858 *nec* L. Agassiz 1847

## *Hymenolepis* and *Drepanodotæna*

### Subgenus 1. *Hymenolepis* sensu Stricto

With the characters of the genus but the hooks have the dorsal root much longer than the ventral and number twenty four to thirty

Proglottis narrow female genitalia ventral to or between testes

### *Hymenolepis* (*Hymenolepis*) *diminuta* Rudolphi 1819

Synonyms—*Tænia diminuta* Rudolphi 1819 *T leptocéphala* Creplin 1825, *T flavopunctata* Weinland 1858 *T varesii* a Parona 1884 *T minima* Grassi 1886

It is a parasite of *Epimys norvegicus* *E musculus* and *F rattus alexandrinus* being not uncommon in rats in Ceylon

The first human specimen was discharged by a child aged nineteen months and was presented to the Boston Medical Improvement

0.2 to 0.5 millimetre in diameter club shaped and has a rudimentary unarmed rostellum and four elliptical suckers. The segments measure 0.66 millimetre in length by 0.35 millimetres in breadth. The anterior proglottides show a yellow spot (hence the name *T flavopunctata*) caused by the distended receptaculum seminis. The posterior proglottides show a brownish grey colour due to the mature uterus. The genital pore is situated laterally. The eggs are oval.

**I**  
cys  
(4  
beetles *Ictis spinosa* and *Scaurus striatus* it is not likely that rat fleas are important as the cysticercus has been found in *Ceratophyllus fasciatus* by Nicoll and Minchin and Johnston in Australia has found it in *Xenopsylla cheopis*

The infection in man is mostly among infants and children

**Hymenolepis (Hymenolepis) nana** von Siebold, 1852

Synonyms.—*Tania murina* Dujardin, 1845 *nec* Gmelin 1790  
*T nana* von Siebold 1852, *nec* van Beneden 1867, *T aegyptiaca*  
 Bilharz 1852, *Diplacanthus nanus* Weinland, 1858

This is the dwarf tapeworm of man and was discovered by Bilharz in the ileum of a boy who died of meningitis in Cairo. It is found in Egypt, Europe, North and South America, Siam and

and mice. Probably it is distinct from this parasite because Grassi

1 in man measuring  
 0.5 to 0.7 millimetre

in breadth

The head is globular and provided with a rostellum with one ring of twenty-four to thirty hooks which are very small, 14 to 18  $\mu$  in length. The neck is relatively long.

There are about 150 proglottides, 0.4 to 0.9 millimetre in breadth and 0.014 to 0.030 millimetre in length. The genital pore is marginal. The mature uterus contains about thirty or more eggs, which are oval and measure 30 to 48  $\mu$  and contain oncospheres measuring 16 to 19  $\mu$  in diameter.

**Life-History**—It is believed that the cysticercus will be found in some insect.

The nearly related *T nana* var *fraterna* completes its development entirely in the rat, for the cysticercus develops in the villus while

be overlooked  
 institutions and  
 occurs in small

numbers, no symptoms result, but in considerable numbers disturbance may occur, which has been assigned by Mingazzini to the effects of toxins.

**Treatment**.—Male fern is the best treatment (*vide* Chapter LXXV)

*nana* and *T. nana*  
 will be necessary to  
 rats and mice. More  
 spreads in crowded  
 fed child should be

insisted upon

**Subgenus 2. Drepanidotaenia** Raulhet, 1892

With the characters of the genus, but the hooks have a dorsal root much larger than the ventral, and are only eight to twenty in number. Proglottids broad, testes three, alongside which lies female genitalia.

Hymenolepis (*Drepanidotezonia*) lanceolata Blochmann 1782

lopes oval (50 by 35  $\mu$ )

Life-History—The cysticercoid lives in a cyclops which is eaten by ducks and geese

SUBFAMILY 2 DAVAININE Braun 1900

Definition—*Tænia* Rostellum and suckers armed Eggs mostly encapsuled

Genus—*Davainea* R Blanchard

*Davainea* R Blanchard 1891

Definition—*Davaineinæ* with a globular head armed with two rings of hammer like hooks and four suckers surrounded by several rings of hooks

Species.—*D. madagascariensis* *D. asiatica*

*Davainea madagascariensis* Davaine 1869

Synonyms—*Tænia madagascariensis* Davaine 1869 *T. demarensis* Daniels 1895

This worm was first discovered by Grenet in children at Mayotte (Comores). Over ten infections have been recorded by Davaine from the Comoro Islands Leuckart from Siam Daniels from

Genitalia number from 500 to 700 being 2 millimetres long by 1.4 millimetres broad. Genital pores are unilateral and near the proximal corner. Testes number about fifty with a long vas deferens and a fusiform corpus pouch. The receptaculum seminis is long and broad. The

Life-History—The life-history is unknown but it is suggested

D	Order	Family	Genus	Species	
Bursata	Strongylidae		<i>Triodontophorus</i>	34 T deminutus	
			<i>Gyrophagostomum</i>	{ 35 C brumpti 36 C stephanostomum var thomasi	
	Metastrongylidae		<i>Metastrongylus</i>	37 M apra	
			<i>Nematodirus</i>	38 N gibsoni	
	Trichostrongylidae		<i>Hylamonchus</i>	39 H contortus	
			<i>Trichostrongylus</i>	{ 40 T colubriformis 41 T probolurus 42 T vitrinus 43 T orientalis	
				<i>Ancylostoma</i>	{ 44 A duodenale 45 A ceylanicum
					<i>Ancylostoma</i>

It is not possible at the present moment to give a scientific and easily workable classification of the Nematoda because the type genera require revision. We group the various families under the following divisions —

- A *Nematoda* in which the bursa copulatrix is merely a cuticular expansion not supported by true fleshy rays in the males—*Non Bursata*
- B *Nematoda* with true bursa copulatrix—i.e. supported by fleshy rays in the males—*Bursata*

### DIVISION NON BURSATA

Leiper gives the following table for differentiation of the non bursate families which are represented in the parasitic nematodes of man —

- A *Gyrophagus* a simple cellular tube—*Trichosomidae*
- B *Cytophagus* a simple muscular bulb —
- (a) Mouth surrounded by several protuberances. Male with one spicule. female with one uterus—*Eustrongylidae*
- (b) Mouth guarded by large fleshy jaws. Male with two spicules. female with two uteri —
- (1) Jaws two in number. skin smooth—*Physaloteridae*  
Jaws two in number. skin spinous—*Gnathostomidae*
- (2) Jaws three in number. skin striated—*Ascaridae*
- (c) Mouth a simple pore without buccal capsule —
- (1) Female without vagina. embryos striated. Male with two equal spicules—*Dracunculidae*





- (2) Female with vagina opening near mouth and convergent embryos not striated. Male with spicules dissimilar—*Filaria*
- (3) Female with vagina in posterior third of body, uterus divergent. Parasitic parthenogenetic adult of *Strongyloides* [1 or free living adult sex (1)]

C. Diophaous with a double muscular bulb—

- (1) Skin smooth, male with two spicules and accessory piece, female viviparous—Free-living adult of *Anisostomidae*
- (2) Skin deeply striated, male with one pelvic female oviparous—*Oxyuris*

EGGS OF DOUBTFUL IDENTIFICATION



Fig. 263



Fig. 264

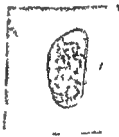


Fig. 265



Fig. 266

FIGS 263-66—EGGS OF DOUBTFUL IDENTIFICATION FROM HUMAN FÆCES

(From preparations and photographs by J. J. Bell)

FIGS 263 AND 265—ABNORMAL EGGS PROBABLY OF *Ascaris lumbricoides*

## NEMATODE EGGS



FIG 255 — *Oxyuris  
vermifilis* EGG  
(X250)



FIG 256 — *Trichouris  
trichura* EGG  
(X250)



FIG 257 — *Trichouris  
trichura* EGG WITH LARVA  
(X250)



FIG 258 — *Ascaris lumbricoides* EGG (X250)



FIG 259 — *A. lumbricoides* EGG SEGMENT  
INTO (X250)



FIG 260 — *A. lumbricoides* EGG UNFERTILIZED



FIG 261 — *Ankylostoma  
duodenale* EGG (X250)

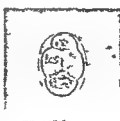


FIG 262 — *Ankylostoma  
duodenale* EGG SEGMENT  
INTO (X250)

FIGS 255-262 — NEMATODE EGGS FROM HILMAN PAPERS  
(From photographs by J. J. Bell)

**Pathogenicity.**—The specimens were found in the papules, and also in the blood at the beginning of the illness, but not in the faces, urine, or sputum

**Life-History.**—Unknown.

#### Leptodera Dujardin 1845

Anguillulidæ with mouth guarded by two three, or six lips. Male with or without bursa which when present never surrounds the point of the tail. Two equal spicules and three pre-anal papillæ. Female with the tail prolonged into a long unsymmetrical point

#### Leptodera pello Schneider, 1866.

**Synonyms.**—*Leptodera pello* Schneider, 1866, *Rhabditis genitalis* Scheider 1880

there Oerley has shown experimentally that they will live in the  
of a mouse

### FAMILY 3 ANGIOSTOMIDÆ.

Nematoda characterized by heterogony each species having a free living bisexual rhabdite form and a parasitic hermaphrodite filarial form

#### Strongyloides Grassi, 1879

**Synonyms.**—*Pseudorhabditis* Perroncito 1881, *Rhabdonema* Leuckart, 1882

The parasitic form has an unarmed mouth long cylindrical œsophagus, which reaches nearly to the middle of the body. The free living stage has a small mouth short œsophagus with a double dilatation, in the hinder part of which are small teeth. The male spicules are of equal size

#### Strongyloides stercoralis Bavay 1877

**Synonyms.**—*Anguillula intestinalis et stercoralis* Bavay, 1877, *Strongyloides stercoralis* 1883.

in faeces, was first described in 1876 by Norman in the faeces and the intestine of French soldiers just returned to Toulon from Cochin China

At first it was thought that there were two species—one in the faeces (*Anguillula stercoralis*) and the other in the bowel (*A. intes*)

## NON BURSATA

## FAMILY 2 ANGUILLULIDÆ

Very small Nematoda mostly free-living rarely parasitic, with

in the middle of the body

Genera.—(1) *Anguillula* (2) *Anguillulina* (3) *Rhabditis* (4) *Leptodera*

*Anguillula* Ehrenberg 1820

Anguillulidæ with small mouth œsophagus with two dilatation of which the posterior has valves. Male without bursa spicules with accessory pieces feather shaped. Female with the vulva in the hinder portion of the body, uterus asymmetrical

*Anguillula aceti* Muller 1785

*Anguillula aceti* is the common vinegar eel which has several times been reported as occurring in the human bladder but the method of infection is unknown

Morphology.—Cuticle not striated body cylindrical tapering a little to the anterior but considerably to the posterior end. Male 1 to 2 millimetres

*Anguillulina* Gervais and Van Beneden 1859

Synonym.—*Tylenchus* Bastian 1864

Anguillulidæ possessing a spine on the oral cavity. Male bursa without papilla uterus asymmetrical

Species — *Anguillulina putrefaciens* Kuhn 1879

*Anguillulina putrefaciens* Kuhn 1879

Synonyma.—*Tylenchus putrefaciens* Kuhn 1879 *Trichina contorta* Botkin 1883

This small nematode lives in onions and in other varieties may at times find access to the stomach with the food and be rejected by vomiting as reported by Botkin in 1883

*Rhabditis* Dujardin 1845

Small Anguillulidæ with no teeth in the oral cavity with accessory pieces to the two male spicules and without lateral ridges

*Rhabditis niellyi* Blanchard 1885

Synonym — *Leptodera niellyi* Blanchard 1885

This parasite was described by Nielly in 1882 in a boy who suffered from an itching papular eruption in Brest, which he had never left

Morphology.—The parasites measured 0.33 by 0.03 millimetre in width and possessed a cuticle with delicate transverse striation a double-bulbed œsophagus, and an intestine but no genital organs

**Gnathostoma Owen, 1837**

*Gnathostoma* Owen, 1837, *Philosophical Transactions of the Royal Society of London*, vol. 27, p. 111, pl. 1, fig. 1.

**Gnathostoma splinigerum Owen, 1837.**

**Synonyms.**—*Cheiracanthus siamensis* Levinsen, 1889, *Gnathostoma siamense* Levinsen 1889, *Cheiracanthus robustum* Diesing, 1839

**Remarks.**—Only two specimens are known one a female, which was obtained by Deuntzer from a young Siamese woman in whose breast a hard painful swelling had formed accompanied with slight fever, and another a male described by Leiper. Nodules the size of beans appeared in the skin from one of which the worms were extracted. Two other similar cases have recently been reported. Leiper has recently compared a male specimen from man with typical specimens of *G. splinigerum* of the tiger, and declares them to be identical.

leads to the cloaca

which the anus opened

**Habitat.**—Subcutaneous tumours in man in Siam. Allied species live in the stomach of pigs and oxen. The species is said to occur in the parish dogs of Calcutta.

**Pathogenicity.**—Man is apparently an aberrant host, for in man only do the worms wander into the connective tissue and form subcutaneous tumours. The species normally lives in the stomach of animals, causing fibrous thickenings.

**FAMILY 5. PHYSALOPTERIDÆ****Physaloptera Rudolphi 1819**

Physalopteridæ possessing mouth with usually two lips each with papillæ and teeth. Posterior end of the male lancet-shaped owing

*isnalis*)—but Leuckart showed that they were but succeeding stages of one life-cycle. It is found in Europe, Africa, India, Ceylon, Indo-China, China, the Philippines, Oceania, the United States, the West Indies, and Brazil.

The fully-developed worm is found in the duodenum and jejunum into the mucosa of which it has bored its way deeply.

**Morphology**—The parasitic adult worm is very small—2.2 millimetres

attacks of diarrhoea.

**Life-History**—The eggs are oviposited into the mucosa of the host's intestine and the embryos hatch and find their way into the lumen and are evacuated with the faeces. On reaching water or moist earth these embryos grow into adult male and female forms which conjugate and then the female lays eggs. The eggs produce free living rhabditiform embryos which moult and turn into filariform embryos which have been shown by Mozocchi and van Durne to penetrate the skin not through the hair follicles but through the horny layer into the rete Malpighii and so into the corium. The experiments of Fulleborn and V. Schilling Torgau in infecting tracheotomized dogs or doves with the



FIG. 267.—RHABDITIFORM EMBRYO OF *Strongyloides intestinalis* BAYAN AS FOUND IN HUMAN FÆCES (After LOOS)

route from the skin via the blood stream directly to the bowel can also take place (*vide* the life history of *Ancylostoma duodenale* p. 663). On arrival in the intestine they burrow into Lieberkuhn's follicles and begin to lay their eggs.

**Pathogenicity**—The parasite is generally believed to cause a catarrh of the small intestine though many believe it to be non-pathogenic.

#### FAMILY 4. GNATHOSTOMIDÆ

mammals

Two genera *Gnathostoma* Owen 1836 and *Tanqua*

anal papillæ and unequal spicules. Females longer than males with vulva situate anteriorly near the mouth

**Synonyms**—*Filicaria* Rudolph 1809, *Filaira* Lee 1840, and some misprints

**Type**—*F. martis* Gmelin 1790

**Species**—*F. bancrofti* Cobbold 1877, (?) *F. demarquaysi* Manson 1895

Some years ago the genus *Filaria* contained a large number of species parasitic in man but the following genera have definitely been separated from *Filaria*—*Acanthocheilonema*, *Drofilaria*, *Dracunculus*, *Onchocerca* and *Hamularia*. At the present time there are only three species known to be human parasites which are referred to the genus *Filaria*—viz, *F. bancrofti* Cobbold 1877, *F. o-aris* Manson 1895, and *F. im-rius* Griseb 1888, in addition to the embryonic forms included in the collective group (*Microfilaria*) and the immature forms in the collective group (*Agamofilaria*). There are however a number of spurious or doubtful forms scattered throughout medical literature and described as human parasites which must be eliminated from the genus and these are—

1 *Filaria* (?) *hominis oris* Leidy 1850—This probably belongs to the Vermithudæ and may have been a mermis accidentally taken into the child's mouth while eating say an apple because it had an obtuse posterior

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untly  
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WORM

The uncertain species are—

1 *Filaria Species* (?) Cholodkowsky 1896—Found in whitlow like tumours on the fingers of peasants in the Twer Government

2 *Filaria Species* (?) Prout 1902

to a widening of the cuticle. The ventral aspect is here covered with cuticular plates and there are four pairs of pedunculated external papillæ and a number of sessile internal papillæ and unequal spicules. Female vulva situated anteriorly. Eggs thick shelled and smooth.

*Physaloptera caucasica* von Linstow 1912

the alimentary canal of

Female 7 millimetres

*Physaloptera mordens* Leiper 1907

Leiper has recently described a large number of cases of infection with *Physaloptera* in natives of tropical Africa which differed from *P. caucasica* not only in size but in the length of the spicules in the male.

FAMILY 6 FILARIIDÆ Claus 1885

Synonyms—*Filaridea* Carus 1863 *Filaridea* Leuckart 1876

or less distinct buccal capsule. Oesophagus slender elongated and may be divided into two portions but has no posterior bulb. Mid gut present rectum present anus subterminal.

Males with one or two unequal spicules and a spirally recurved tail provided with papillæ and in some cases with lateral alæ. Females larger than the males with or without a vulva in the gravid worms which when present is situated anteriorly. Uterus

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SUBFAMILY FILARIINÆ Stiles 1907

*Filaria* Muller 1787

Definition—This is doubtful *Filaridea* with long slender filiform bodies, anterior extremity attenuated obtuse posterior very attenuated more so than the head. Cuticle without transverse striation and without bosses. Male shorter than the female with spirally bent tail provided with lateral cuticular alæ pre and post



at night, there is no difficulty for the *Microfilaria* to reach the stomach of a mosquito in the tropics and here and in its thorax

*Panoplitis africanus* in Central Africa

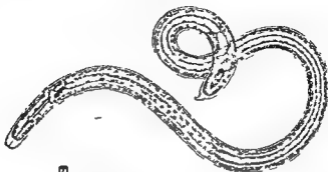


FIG. 272 —LATE STAGE OF THE DEVELOPMENT OF *Filaria bancrofti* COBBOLD IN *Culex fatigans*  
(After LOOSS from Mense's 'Tropenkrankheiten')

Incomplete results have been obtained with *Culex microannu- latus*, *C. albopictus*, *C. lamia*, *C. tritaeniorhynchus*, *C. albimans* and negative results in *C. notoscriptus*, *C. annulirostris*, *C. hispidus*, *C. vigilax*, *C. usgrithorax*, *C. procax*, *A. musivus*, *A. funesta*, *A. maculipennis*, *Pulex serraticeps*, *Stomoxys* (?) *Clinocoris lectularius*



FIG. 273 —DIAGRAM OF THE DEVELOPMENT OF *Filaria bancrofti* COBBOLD IN THE THORAX HEAD AND LABIUM OF A MOSQUITO

The *Microfilaria* enter with the blood into the mosquito's stomach, and there escape from the sheath by rupturing it at the anterior extremity.

They then pierce the wall of the stomach and find their way into the muscles of the thorax where they grow considerably till 1.5 millimetres long and 0.25 millimetre broad.

3 There was a parasite called *Spiroptera hominis* Rudolphi 1819 which appears to have been spurious being really *Filaria communis* of which *Filaria piscium* is a synonym

*Filaria bancrofti* Cobbold 1877

Simonson The 2 ... Cal ... 1877

that its true anatomical habitat is the blood Bancroft in 1876 discovered the adult female form Borne in 1888 discovered the adult male In 1890 Manson discovered that the *Microfilaria* on to the lungs osquitoes as the *Filaria*

escaped from their insect hosts into water and reached man by this means Later Bancroft conjectured that *Filaria* might be inoculated directly into man and about this time sent infected mosquitoes to Manson in London Bancroft's material was investigated at Manson's request by Low who discovered that the *Filaria* migrate to the mouth parts of their insect hosts after a period of growth within the musculature A little later, independently, James made the same discovery in India In the meantime Grassi and No<sup>1</sup> investigated the life-history of a

successful in inoculating normal dogs by means of mosquitoes fed on infected dogs Fulleborn and Bahr have most carefully studied the subject

**Morphology**—*General Characters*—The worms are whitish in colour long and filiform with a smooth cuticle and a globular head terminating in a simple circular unarmed lipless mouth The tail is rounded

*Sexual Characters*—The male is smaller than the female, and a complete s...

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anal and t  
fourteen pairs

with a bulbous cuticular expansion at the tip of the tail. Anterior end 0.25 millimetre in front of the posterior extremity, vulva 0.71 millimetre behind the anterior end.

**Life-History.**—The egg develops into a microfilaria which has no sheath, shows no periodicity, and measures  $230 \mu$  by  $5 \mu$ . Its tail is tapering and sharp pointed and it moves actively. Nothing further is known as to the life-history.

**Pathogenicity.**—Nil.

#### *Filaria inermis* Grassi

**Synonyms**—(?) *Hamularia lymphatica* Freutler, (?) *Filaria palpebralis* Pavesi 1867 (?) *Filaria labialis*, etc.

Several female filarial worms have been described from Southern

differ, however, in the position of the vulva.

#### (*Microfilaria*) *Le Dantec* 1904

A collective group of the larvae of unknown adult *Filaridæ* found in the blood of man and other vertebrates.

#### (*Microfilaria*) *powelli* Penel 1905

This microfilaria was found by Powell in 1903 in the blood of a Bombay policeman.

It showed a nocturnal periodicity (?) was provided with a sheath measured  $131 \mu$  by  $5.3 \mu$  and had a truncated tail.

#### (*Microfilaria*) *philippinensis* Ashburn and Craig 1906

Described from the blood of a man in May 1906. It measures  $131 \mu$  by  $5.3 \mu$ . It moves actively and is provided with a sheath, within which it is clearly seen at the anterior end. The serrated prepuce and the anterior V-spot situate the outer coat by its

They also undergo development obtaining an alimentary canal and a three lobed tail. When so far developed they leave the thorax and pass through the prothorax and head into the labium where they remain until the mosquito bites a man when they escape into the skin by making their way through Dutton's membrane which is a thin membrane between the labella and the chitinous skeleton of the labium as was demonstrated by Lebrede (For further details see Chapter LXXI)

Their further development in man is not known but in due course they become adults.

**Pathogenicity**—Usually non pathogenic these worms under certain circumstances can cause elephantoid fever elephantiasis lymph scrotum etc or in other words filariasis.

#### *Filaria taniguchi* Penel 1905

This *Filaria* was found in a lymphatic gland from a person living in Ama kusha in Japan. Only the female and the microfilaria are known.

The former measured 68 millimetres in length and 0.2 milli-

mouth.

The latter measured  $16.4 \mu$  long by  $8 \mu$  broad possessed a sheath and a truncated tail and showed in its internal periodicity. Ieper regards this species as the same as *I. bancrofti*.

**Life-History**—Not known.

**Pathogenicity**—Not known.

#### *Filaria ozzardi* Manson 1897

**Synonyms**—*Filaria ozzardi* Manson 1897 *F. juncea* Railliet 1908.

In 1895 Manson discovered a microfilaria in blood films from natives of St Vincent in the West Indies which he named *Filaria demarquay* after the discoverer of the microfilaria of *F. bancrofti*. In 1897 he found in blood films from Carib Indians of British Guiana another microfilaria which is the same as that called *I. demarquay* but which he provisionally considered to be a different species and called *I. ozzardi*. The name *demarquay* is preoccupied.

Lately Penel and Manson have come to regard them as identical. Galley found the adult females in the West Indies.

It is found in St Vincent, Dominica, Trinidad, St Lucia in the West Indies and in British Guiana where it is found in jungle districts. The adults live in the connective tissue at the root of the mesentery and elsewhere.

**Morphology**—The male has not yet been described and only a fragment of a posterior end has been found. The female measures 65 to 80 millimetres in length and 0.21 to 0.25 millimetre in breadth.





It does not appear to have the central viscus seen in microfilaria of *F. bancrofti*. In stained specimens there is an area free from nuclei anteriorly a transverse break at  $34\mu$  = V spot at  $49\mu$  and the tail spot at  $125\mu$ .

A short type measuring 90 to  $100\mu$  has been noted. There

history. Its larvae are said to have been found in the thorax of *Panoptes (africanus?)*, *Stegomyia fasciata* and *Tan oryzae* *fuscopennatus*. It will not develop in a large number of blood suckers—e.g. species of *Inoplex*, *Culex*, *Pulex*, *Pediculus* and *Urenozenia*. Wellman and Ieldham claim to have found its development in a tick (*Ornithodoros moubata*) as first suggested by Christy. According to Leiper however these are spermatozoa.

It is to be noted that it only occurs in areas covered with dense forest and possessing swamps which indicates that the host probably requires shade in the day and water to lay its eggs in.

**Pathogenicity**—Nil

#### *Dirofilaria* Railliet and Henry 1911

Tylarudæ with very long filiform body with a striated cuticle unprovided with bosses mouth unarmed with six cephalic papillae. Males with a spiral tail. Female with vulva in anterior hundredth of body. Viviparous.

**Type**—*Dirofilaria immitis* Leidy 1856

#### *Dirofilaria* magalhæsi Blanchard 1895

**Synonymy**

In 1887

found by  
child in Rio de Janeiro

For a time it was mistaken for *F. bancrofti* but in 1894 Marcy pointed out that it was a different species and in 1895 Blanchard gave it the present name.

**Morphology**—The worms were white opalescent and transversely striated the head club shaped and simple mouth terminal oesophagus with a bulb and there was a rounded tail. The male measured 83 millimetres in length and 0.08 to 0.4 millimetre in breadth and possessed a rounded tail with a cloaca 0.11 millimetre from its tip with two spicules and four pre anal and three post anal pairs of papillae.

head 0.6 to 0.8 millimetre behind the tip of

the tail

**Life-History and Pathogenicity**—Unknown

Type—*Acanthocheilonema dracunculoides* Cobbold 1870

Other species are *A. perstans* Manson 1891 (*A. recondita* Grassi 1890 and *A. grassii* Noë 1907)

### *Acanthocheilonema perstans* Manson 1891

blood of negroes from the Congo Daniels found the adults in British Guiana The geographical distribution known at present is Tropical Africa and British Guiana

**Morphology**—The adults are found as a rule free in the connective tissue at the base of the mesentery around the pancreas behind the pericardium and behind the abdominal aorta and the suprarenal capsules The body is cylindrical uniform except towards both ends when it tapers a little



FIG. 274.—EMBRYO OF *Acanthocheilonema perstans* MANSON

The male rarely met with is 45 millimetres in length by 0.6 millimetre broad with a greatly curved tail which ends in a bifid prolongation of the cuticle Low describes two unequal spicules and

two unequal cuticular lobes The mouth is simple and small and the alimentary canal shows no differentiation into oesophagus or intestine The anus opens upon a papilla 0.15 millimetre in front of the tip of the tail The uterus is double and when full of eggs and embryos in various stages of development nearly fills the body The vulva is 0.6 millimetre behind the anterior extremity

**Life-History.**—The egg undergoes its development in the uterus and the microfilaria escapes from its egg membrane and appears



*L. loa* is a parasite of the superficial connective tissue—the conjunctiva the subcutaneous fat and the superficial aponeuroses in all parts of the body. It probably only occurs in man for the Cameroons requires s on the West Coast of ng most common in Old

Calabar the Cameroons and the Ogome River. It is however by no means confined to the coast for it is known to penetrate at least 600 miles into the interior of Africa.

The cases reported fr —  
cases from the West Inc  
been imported from the

The parasite appears to have never become endemic outside a given area which means that the animal by which it is spread has a restricted geographical range.

**Morphology**—The male is a thin white almost transparent



FIG 275—*Loa loa* COBBOLD  
POSTERIOR EXTREMITY OF THE  
MALE



FIG 276—*Loa loa* COBBOLD  
POSTERIOR EXTREMITY OF THE  
FEMALE

(After Looss)

worm with a body tapering to each extremity and measuring 25 to 34 millimetres in length and 0.273 to 0.430 millimetre in breadth. The head is like a truncated cone the neck is but feebly indicated. The tail more or less incurved has a rounded tip from which the anus is distant 74 to 87  $\mu$ . There are three pairs of well marked pre anal and two pairs of post anal papillae with sometimes a little tubercle on each side of the middle line far posteriorly.

The spicules are two in number and unequal and are usually stated to differ but little in length. Penel says that the larger is traversed by a fine canal opening laterally a little distance from the free extremity.

The cuticle consists of a superficial thin translucent layer and a deeper perpendicularly striated layer. Scattered over this cuticle there are rounded thickenings or bosses the smaller being 9 to 11  $\mu$  and the larger 14 to 16  $\mu$  in height. The thickness at the posterior extremity is variable as also is the constriction correspond

**Dirofilaria immitis** Lesdy 1856

This worm lives in the right heart and in the veins of the dog and also of the wolf and the fox in Europe and tropical regions. It is very common in China and Japan.

Power 16 mm

Arab were *Schistosoma hæmatobium* as he carefully reported never mentioning *D. immitis*; Braun however seems to think it possible that this worm may occur in man.

**Morphology**—The worm is long measuring 12 to 18 centimetres by 0.7 to 0.9 millimetres in the male and 25 to 30 centimetre by 1.0 to 1.3 millimetres in the female and filiform with a smooth rounded cuticle and a rounded

in the dog

They enter the Malpighian tubules or their epithelial cells where they moult and grow eventually passing via the body cavity to the labrum.

They escape through Dutton's membrane on to the skin when the mosquito bites and so enter the dog.

**Loa Stiles 1905**

Filaridæ with bosses on the cuticle and with large caudal papillæ

Species—*Loa loa* Guyot 1778

**Loa loa** Guyot 1778

**Synonyms**—*Filaria oculi* Gervais and van Beneden 1859  
*Dracunculus oculi* Diesing 1860 *D. loa* Cobbold 1864 *F. subco-*  
*juicivialis* Guyon 1864

The first

was the first person to record the presence of a worm in the eye. Guyot in 1778 and 1805 thought it was a *Strogylis* and used the term loa for the first time in European literature. In 1891 Manson found a microfilaria in the blood of several negroes from the Congo which differed from those already described and which he named *Filaria diurna* and further suggested that it might be the same as

proved

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esis has been

root Henly

*diurna* are

of the same

from the graceful curves of *M bancrofti*. The anterior V spot can be seen and probably opens at the apex to the exterior as Penel has observed that the stum penetrated easily at that spot probably there is also an opening at the tail spot. In stained specimens the first 8  $\mu$  is clear without nuclei at 62  $\mu$  the column of nuclei is broken by an irregular transverse spot, at 99  $\mu$  by a triangular spot.

The last nuc  
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in the perip

No explana

It has be

life-history

this has been shown to be the case by Jesper who also finds a development in *C sila ea*, but the method of infection of man is unknown

After entering the human body it would appear probable that the worm takes some three to four years to reach sexual maturity and that it is long lived—i.e. fifteen or more years. The reason for believing this is the fact that immature forms may be noted in children and the fully grown worm found in the adult. When the worm dies it may become calcified.

TABLE SHOWING THE DIFFERENCES BETWEEN THE EMBRYOS OF *Filaria bancrofti* AND *Loa loa* (MODIFIED FROM DR G C LOW)

	<i>Filaria bancrofti</i> Embryos	<i>Loa loa</i> Embryos
Average length	0.317 millimetre	0.245 millimetre
Average breadth	0.0034-0.0075 millimetre	0.0075-0.0070 millimetre
Break in cells from head	0.050 millimetre	0.042 millimetre
V spot from head	0.090 millimetre	0.060 millimetre
Eggs (average)	0.020 X 0.033 millimetre	0.042 X 0.033 millimetre
Character of curves in dried specimens on slides	Spiral coils	In a wavy line
Periodicity	In blood at night (or in equal numbers in blood by day and night Fiji etc.)	In blood by day
Periodicity when habits of sleeping and waking changed	Inverted	No change

**Pathogenicity**—It may be noted under the skin of the finger the back the breast the scrotum and penis the eyelid under the conjunctiva the mucosa of the tongue. It moves quickly and

ing to the neck. The viscera are enclosed in a cylindrical musculo-cutaneous tube. The mouth is terminal, small, unarmed, and surrounded by a powerful muscular cone. The oesophagus is short and without a bulb. The intestine opens via the rectum at the anus near the posterior extremity. The excretory pore is 0.65 millimetre from the anterior end of the body.

The genital apparatus consists of a tubular testis and vas deferens

extremity resemble those of the male.

The genital system consists of a vulva situated on a little elevation  $23 \mu$  in height and distant some 2.5 millimetres from the anterior extremity. This vulva leads into a thick-walled canal—the vagina—from which the two uterine tubes, full of embryos and eggs, diverge and end in the ovaries.

122



FIG. 277.—EMBRYO OF *Loa loa* GUYOT

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cent of the population. It has not been recorded as far south as the Congo. There has been recorded three cases from Dutch Guiana and Leiper has recently confirmed its occurrence in Guatemala where according to Robles the infection is very common.

**Morphology**—The male worm has a white filiform body slightly attenuated at the ends covered with a transversely striated cuticle measuring 30 to 35 millimetres in length and 0.14 millimetre in breadth.

The head is rounded the mouth unarmed the alimentary canal straight and undifferentiated the anus subterminal 0.49 millimetre in front of the posterior end. Other canals are reported which probably belong to the generative and excretory systems.

The tail is strongly recurved and somewhat flattened on its concave aspect. Brumpt reports three pairs of papillae on each side of the anus and three pairs of post anal papillae. There are two unequal spicules the larger 177  $\mu$  and the smaller 82  $\mu$  in length.



FIG 78—MICROFILARIA OF *O. chocerca solivius* LEUCKART  
(After I. Leibern)

The female  
70 millimet  
cuticular st  
recurved

is 0.76 millimetre from the anterior end

**Life-History**—The worm is said to lie in a lymphatic which becomes inflamed and a perilymphangitis causes a condensation of connective tissue in which males and females are embedded leaving the posterior end of the male and the anterior end of the female free in the lymph space. The embryos pass out of the uterus into this space where they can be found 250 to 300  $\mu$  in length and 5 to 6  $\mu$  in breadth with a rounded head and a body which tapers during the last fifth and terminates in a pointed tail. There is no sheath. The anterior V spot is clearly seen. The microfilaria have not been seen in the blood and their further development is quite unknown.

1 Brumpt  
tsetse



which indicated that they were not *A. lumbricoides*. On further examination they were found to be

from *A. lumbricoides*. He therefore formed two new genera—*Toxascaris* and *Belascaris*. So far, *Toxascaris* has only been recorded once in man.

**Morphology.**—Body white or reddish, head curved dorsally with two membranous lateral expansions broader behind than in front. Male 5 to 10 centimetres in length with curved tail possessing two small lateral membranous wings and twenty six papillæ. Female 9 to 12 centimetres in length with an obtuse tail. Eggs 75 to 80  $\mu$  in diameter.

**Life-History.**—Development is similar to *A. lumbricoides*.

**Pathogenicity.**—It often causes intestinal and nerve symptoms in dogs. Post mortem the mucosa of the intestine is tumefied and catarrhal.

#### *Belascaris Leiper 1907*

**Definition.**—Ascaridæ with the anterior end of the body bent ventrally, cuticle coarsely striated, œsophagus with a distinct bulbous portion. Male with a probular tail—*i.e.* like a closed fist with forefinger semi-extended. Immediately behind the anus there is a protuberance with a pair of papillæ. On the tail there are two ventral and two lateral pairs of papillæ the tips of which support a slight expansion of the cuticle. The testis is situated in the anterior half of the body. The vesicula seminalis is remarkably long and there is a short ejaculatory duct. Female with vulva situated in the anterior part of the body. Egg with a honey-combed shell.

**Type.**—*Belascaris cati* Schrank 1788

#### *Belascaris cati* Schrank 1788

**Synonyms.**—*Fusaria mystax* Zeder 1800 *Ascaris alata* Bellingham 1839

This *Ascaris* is common in cats and has been recorded nine times in man.

**Morphology.**—Head curved ventrally with two membranous lateral expansions. Male 4 to 6 centimetres in length, female

**Pathogenicity**—Lymphangitis perilymphangitis sometimes acute and with fever and resulting in small tumours are their pathological signs

FAMILY 7 DRACUNCULIDÆ LEIPER 1912

*Dracunculus kniphofi* 1759

Nematodes with small males and long females. In the latter a vagina is wholly absent the embryos being discharged by rupture of the gravid female

*Dracunculus medinensis* (Linnaeus 1758)

**Synonyms**—*Dracunculus telurus* Velsch 1874 *Dracunculus medinensis* Vol. 1, p. 26

*Dracunculus* Beinwurm Brichwasserswurm tankworm or dragon new is endemic in tropical Africa India Persia Turkestan Arabia and some places in South America to which it was imported from Africa



FIG 279—*Dracunculus medinensis* LINNAEUS

It has been known since the most remote periods and it was probably the fiery serpent mentioned by Moses who apparently knew the method of twisting the worm out on a stick as he apparently made a model of this method of extraction. Galen called the disease caused by these worms dracontia.

The anatomy of the worm was carefully studied by Bastian in 1863 and the infection of *Cyclops* with the larva was observed by Fedchenko in 1870 and confirmed later by Manson. Charles found a calcified guinea worm in 1890 which he described as the male.

Leiper 1907 has repeated these experiments and has further proved that when monkeys are fed on infected *Cyclops* the males and females can be found.

**Morphology**—The female is a long white filiform worm 50 to 100 millimetres in diameter.

popular  
consideral





FIG 283—*Oxyuris vermicularis*  
(LINNÆUS 1767) MALE

(From a photograph by J J Bell)



FIG 284—*Oxyuris vermicularis*  
(LINNÆUS 1767) FEMALE



FIG 285—*Oxyuris vermicularis*  
(LINNÆUS 1767) HEAD  
(From a photograph by J J Bell)



FIG 286—*Oxyuris vermicularis*  
The male is to the left the female  
to the right  
(After Claus)

**Genus Lagochilascaris Leiper, 1909.**

**Definition.**—*Ascaridae* with dense cuticular lips and interlabia

**Lagochilascaris minor Leiper 1910**

This species has been found in the pus of subcutaneous abscess in man in Trinidad

**Morphology.**—Males 9 millimetres in length by 0.5 millimetre in breadth, with bent posterior part of body. Female 15 millimetres in length, straight posteriorly. The vulva with two projecting lips opens 6 millimetres from the anterior end

**Hosts.**—Possibly one of the Carnivora. Man is an accidental host

**Habitat.**—Probably intestinal in its normal host. The specimens were found in abscesses under the skin in man. An allied species, *A. major* occurs in the intestine of the lion in East Africa

**FAMILY 10 OXYURIDÆ Dujardin**

**Genus Oxyuris Rudolphi 1803**

**Definition.**—*Nematoda* in which the three labial papillæ are not very distinct. Oesophagus long, with a double dilatation. Skin markedly striated. Male with curved posterior end, one spicule, and two pairs of pre-anal papillæ. Female with straight posterior end which tapers to a point. Vulva in the anterior part of the body

**Type.**—*Oxyuris vermicularis* Linnæus 1767

**Oxyuris vermicularis Linnæus 1767**

**Synonyms.**—*Iscaris vermicularis* Linnæus 1767 *Fusaria ver*

tail  
 Life-His  
 50 to 52 µ  
 embryo  
 ventral

***Triodontophorus* Looss 1901**

**Synonyms**—*Triodontus* Looss 1901 *ne* Westwood 1845 *Terridens* Railliet and Henry 1909

Strongylidæ with small almost spherical thick walled oral cavity arising from the floor of which three teeth are found each of which consists of two surfaces joined together at an acute angle. Male bursa is finely serrated at the edge. The female genital orifice is situate a short distance in front of the tip of the tail.

Found in horses and men

***Triodontophorus deminutus* Railliet and Henry 1905**

**Synonym**—*Termodens deminutus* (Railliet and Henry 1905)

Railliet and Henry in 1905 discovered that a male and female parasite presented in 1865 by Monestier to the Paris Natural History Museum and collected post mortem from an American negro who died in Wyotte were not ankylostomes but belonged to Looss's genus *Triodontophorus*. They named them *deminutus* because of their small size. Leiper also met with some specimens of the same species collected in Nyassaland and Iorenço Marique. It has now been recorded in a number of cases. It is also found in monkeys. Their normal habitat is the large intestine.

**Morphology**—To the naked eye their size and general appearance resemble those of ankylostomes. The body is white cylindrical tapering towards

ing into the lumen which are continued forwards as three stout unguis prongs or teeth one dorsal and two ventral. The characters of these teeth

**Life-History and Pathogenicity**—Unknown

duced into the mouth or nose by self infection fruits vegetables etc hatch and grow in the small intestine into male and females

After fertilization the males die off and the females travel into the cæcum and later into the colon and have a great tendency to



FIG 287—DEVELOPMENT OF *Oxyuris vermicularis*  
(After Leuckart from Stiles)

wander through the anus to the outside The duration of life of *O. vermicularis* in the human body is not known

**Pathogenicity**—In large numbers may set up enterocolitis in small numbers it causes only irritation

## DIVISION BURSATA

**Definition**—Nematoda with true cuticular bursa in the male

**Classification**—Leiper gives the following table for the differentiation of the various bursate families represented by species in man —

A Mouth with simple pore no buccal capsule —

(a) Male with large bursa and elongated rays female oviparous uteri divergent ovjectors present Intestinal parasites—*Trichostrongylidæ*

(b) Male with short bursa and tufted rays female oviparous uteri convergent ovjectors absent Lung parasites—*Metastrongylidæ*

B Mouth a simple opening leading into a large buccal capsule guarded by chitinous structures

(1) Mouth capsule terminal guarded by ring of setae—*Strongylidæ*

(2) Mouth capsule terminal guarded by ring of setae—*Strongylidæ*

## BURSATA

### FAMILY II STRONGYLIDÆ Cobbold 1864

Nematoda with cylindroid rarely filiform bodies mouth usually with armature and in Male with a bursa with two ovaries

vulva situated medially or posteriorly As a rule the species are small Oviparous

**Type Genus**—*Strongylus* Mueller 1780

**Other Genera**—*Tridontophorus* Looss 1891 *Gyophagesomum* Molin 1860

## FAMILY 12 METASTRONGYLIDÆ Leiper, 1912

Nematoda with simple mouth very small cavity and not enlarged to form a buccal capsule and a bursa in the male with true but stunted rays. Two spicules.

This family includes a number of genera parasitic in the lungs of domesticated animals.

*Metastrongylus* Leiper 1908

*Metastrongylus* Molin 1861

*Metastrongylus apri* Gmelin 1789

✓ in Klausenberg in  
 ■ looked upon as  
 cause pneumonia and

**Morphology.**—Body relatively short colour white or brown mouth with six lips of which the two lateral are the largest. Male 12 to 25 millimetres in length with bilobed bursa with five ribs in each lobe and thin spicules. Female 15 to 20 millimetres in length with a

years ago as it may be natural for the worms at times to migrate on to the buccal like the *Ankylostoma*. It is interesting to note that Leuckart

## FAMILY 13 TRICHOSTRONGYLIDÆ Leiper 1912

Nematoc  
 versely or  
 armature,  
 posterior half of body ovjectors present oviparous,  
 parasites

*Nematodirus* Ransom 1907

Trichostrongylidæ with bursa provided with double ventral median ribs two dorsal ribs spicules long and filiform without accessory piece. Head 50 microns in diameter, cuticle with eighteen distinct longitudinal ridges. Cervical papillæ absent

**Œsophagostomum Mohn 1861**

Strongylidae in which the mouth is small and circular with a chitinous ring which carries a fold and six papillae. There is a

**Species.**—*Œsophagostomum aplostomum* and (*Œsophagostomum* var *thomasi*) Railliet and Henry 1909 are known in man but other species are found in the gorilla chimpanzee and other monkeys

**Œsophagostomum aplostomum Willach**

**Synonym**—*Œsophagostomum brumpti* Railliet and Henry 1905

This worm was found by Brumpt in cyst like nodules in the caecum and colon of a negro on the Omo River near Rudolph's Sea in East Africa. Found also in monkeys

According to Leiper this parasite occurs in 5 per cent of the natives of Northern Nigeria

**Morphology**—Form 1 a c

**Life-History**—Outside the body similar to that of the Ankylostomes

**Pathogenicity**—The young form cyst like nodules in the large intestine

**Œsophagostomum stephanostomum var thomasi Railliet and Henry 1909**

This species was found by Wolferton Thomas in tumours numbering 187 in the large and small intestines of a man in Brazil. The tumours were in some instances large enough to diminish the lumen of the bowel. Form 2 a

male and a female ch 1904 was found (*Œ. stephanostomum*) d in the chimpanzee

**Morphology**—Body cylindrical thick only attenuated towards the extremities. Buccal capsule with an external crown of 18 lamellae. Male 17 to 22 m l. Female 15 to 20 m l. 5 to 10

**Life-History**—Not known

**Pathogenicity**—Forms tumours in the wall of the small and large intestines

### Trichostrongylus Looss 1905

Trichostrongylidae with body tapering gradually from the genital opening anteriorly. Head with three small lips and blunt or pointed papillae without cuticular protuberances or neck papillae. Cuticle transversely striated. Oesophagus long. Male with the bursa closed round by large side flaps without evident median folds, spicules spoon-like with a boat-shaped accessory piece. Female with the genital opening in the posterior half of the body. Tail short with two small papillae near the tip. Egg thin-shelled.

Type Species.—*T. colubriformis* Looss

Four species of

*colubriformis* T. pr

### Trichostrongylus colubriformis Giles 1892

Synonyms—*Strongylus colubriformis* Giles 1892 *S. instabilis* Railliet 1893 *S. subtilis* Looss 1895

This species was found by Looss in post mortems on fellows in Alexandria and Cairo and also by Ijima in a woman in Japan. In man however it is only an accidental parasite being usually found in the duodenum rarely in the stomach of sheep, antelopes, dromedaries in Egypt, monkeys in North America and sheep in India.

Morphology—Male 4 to 5 millimetres in length and 0.08 millimetre in thickness just in front of the bursa. Spicule 135 to 145  $\mu$  in length and the accessory portion 70  $\mu$  in length with a long muscular oesophagus. Bursa with two lateral semicircular wings connected by a cross bridge. Ribs arranged asymmetrically. Female 5 to 6 millimetres in length and about 0.09 millimetre in breadth. Posterior extremity tapers to a pointed tail in front of which the anus is situated 0.055 to 0.07 millimetre and the vulva 1.05 to 1.2 millimetres. Eggs 72 to 80  $\mu$  by 40 to 43  $\mu$  when oviposited generally contain eight to twelve celled embryos.

Life-History—Resembles that of *Ancylostoma duodenale* outside the body according to Leiper.

Pathogenicity—Believed to be unimportant.

### Trichostrongylus orientalis

1895

found in the faeces of a population of certain

districts in Japan and is probably the same as that recorded

### Trichostrongylus probolurus Railliet 1896

Synonym—*Strongylus probolurus* Railliet 1896

This parasite lives in the duodenum of sheep, antelopes and dromedaries in Egypt and has also been found in man in Egypt.





capsule wall. The aperture of the dorsal oesophageal gland can be seen in the dorsal wall of the mouth capsule.

Male with a three lobed bursa broader than long with two spicules. Female with vulva behind the middle of the body.

Species—A number of species are known in man, dogs, bears, civet cats and other carnivora.

***Ancylostoma duodenale* Dubini 1843.**

Synonyms—*Strongylus quadridentatus* von Siebold 1831. *Dochmus ancylostomum* Molin 1860. *Sclerostoma duodenale* Cobbold. *Strongylus duodenale* Schneider 1866. *Dochmus duodenalis* Leuckart 1876 *pro parte*.



FIG 288 — *Ancylostoma duodenale* DUBINI 1843  
MALE

FIG 289 — *Ancylostoma duodenale* DUBINI  
1843 MALE BURSA

(From photographs by J. J. Bell)

the *Harita Samhita* under the term *pandu roga* which was said to be caused by swallowing clay. If this is correct then knowledge of anaemia due to geophagy is very old indeed. If the ancients did really know about this worm and the disease caused thereby the knowledge was totally lost and it was not till Dubini in 1838 discovered the worm in a peasant woman in Milan that modern medicine knew anything about its existence. Pruner in 1846 found the parasites in Egypt and Griesinger in 1851 showed

Life-History — This is unknown

Pathogenicity — Believed to be unimportant

*Trichostrongylus vitrinus* Looss 1905

*T. vitrinus* is found in the duodenum of sheep and dromedaries

FAMILY 14 ANCYLOSTOMINÆ LOOSS 1911

Strongyles with armed mouth and bursa copulatrix provided with ribs

Subfamilies — Ancylostominae and Bunostominae

SUBFAMILY ANCYLOSTOMINÆ LOOSS 1911

Ancylostomidae with more or less funnel shaped mouth capsule its walls on the ventral side and especially towards the anterior edge provided on each side with two longitudinal thickenings projecting outwards like ridges. In the gutter like depressions between the ridges lie the terminations of the dorsal and lateral papillary nerves. Floor of the mouth cavity with one pair of inner ventral teeth otherwise free from tooth like structures. Aperture of the dorsal œsophageal gland situated in the wall of the mouth capsule. Bursa of the male closed all round. Only one third of the dorsal ray is cleft. The course of the genital tubes is longitudinal.

Type Genus — *Ancylostoma* Dubini 1843 Other genus *Lucina* Gröblich 1789

*Ancylostoma Dubini* 1843

Synonym *T. vitrinus* Looss

While the bursae are closed all round the longitudinal rib like thickenings of the external surface of the

head gland, which runs through nearly half the length of the body. In the floor of the mouth there are two ventral chitinous plates and the prominent opening of the dorsal head gland (often called a tooth).

Male measures about 10 millimetres in length by 0.4 to 0.5 millimetre in breadth and possesses a bursa copulatrix at the posterior end which is umbrella shaped and supported by chitinous rods which are arranged as follows. In the median dorsal line is the costa dorsalis which divides dorsally into two small branches which are ramified at their tips. Postero laterally there is one root on each side—the single costa dorsalis externa in front of which is a single broad lateral root which divides into the costa lateralis posterior the costa lateralis media and the costa lateralis externa while anteriorly also on each side is the costa ventralis. Through the opening of this bursa project the two spicules unless they are retracted which are long and slender and measure about 2 millimetres in length. The male generative apparatus consists of a testis in the form of a tube an oval vesicula seminalis and a long cement gland whose secretion fixes the male to the female during conjugation and a spicule sac.

The female measures 12 to 13 millimetres in length and has the vulva at the junction of the middle and hinder parts of the body from which a short vagina opens into two tubes which are divisible into ovijector uterus receptaculum seminis and an ovary.

**Life-History**—The adult worms live chiefly in the

jejunum of the host where they feed upon the villi. Blood is only accidentally found in a worm. Here the females lay the eggs which are oval in form with broad rounded poles surrounded by a colourless shell which is really double but looks single inside which lies an oval granular mass separated from the shell by a considerable space. As the



FIG 201—Anterior end of *Acylina duodenale* DUBINI MALE (After Looss)



FIG 202—ANTERIOR END OF *Acylina duodenale* DUBINI (After Looss from Mensen & Tropicankranken)

egg travels down the alimentary canal the granular mass divides into two and finally into four segments in which condition the egg is usually found in the faeces. Development proceeds in the faeces depending upon the temperature of the atmospheric air. In twenty four hours if supplied with air water and heat the embryo

that it was the cause of Egyptian anæmia Wucherer in 1872 found that it was the cause of tropical anæmia in Brazil (called oppilação) Perroncito found that it was the cause of the anæmia

causes is slowly being realized and the deaths from anæmia general dropsy and so called beri beri etc in different tropical regions are being found to be due to this animal

The development and method of infection have been completely traced out by Looss in Egypt in a most masterly manner

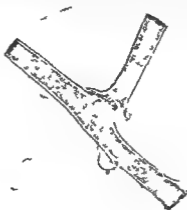


FIG. 290.—COPULATION OF ANCYLOSTOMA  
(from a photograph by J. J. Bell)

In 1902 Stiles found that under the term *Ancylostoma* two different parasites were being confused one corresponding to DuRoi's *Ancylostoma duodenale* and the other new which he named *Ancylostoma braziliense*. The latter is now widespread this latter

is probably not fully known with *Vecator*. It is supposed to be found in mines and regions and in mines and

tunnels in colder climates in which of course the air temperature is higher than that of the outside

**Morphology**—The body is cylindrical tapering from back to front in both sexes. During life it is flesh coloured. The cuticle is ringed. The mouth is terminal with a chitinous wall which ventrally carries two pairs of hook like teeth and dorsally one pair. Close to the base of the outer ventral tooth opens the single-celled

letter many are 1102 - 45 1 - 25 4

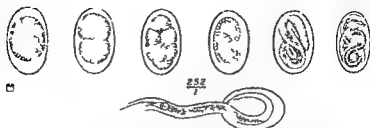


FIG 295 — *Ancylostoma duodenale* DUBINI DEVELOPMENT OF THE RHABDITIFORM EMBRYO  
(After Looss except the last figure which is after Ferroncito)

the intestine. The time occupied by this journey is believed to be from seven to ten days. In the skin they undergo their second ecdysis and later a third and fourth ecdysis takes place in the alimentary canal, the third in four to five days and the fourth from four to six days after their arrival. They now measure 3 to 5 millimetres in

length and eight days later the generative organs begin to attain maturity and the first copulations take place and a few days later the first eggs appear in the faeces thus completing the cycle of development of which the portion after infection occupies four to six weeks.

The more important morphological changes which take place in the human body may be briefly recapitulated.

On entering the skin the third stage of development begins during which the provisional buccal capsule is formed. A third ecdysis ushers in the fourth stage characterized by the provisional buccal capsule armed

with a dorsal and a ventral pair of teeth. During this stage the sexes become differentiated and the permanent buccal capsule is formed. The fourth ecdysis results in the appearance of the adult worms.

The number of females can be calculated from the number of eggs in the faeces by the formula  $X = \frac{A}{47}$  where  $X$  is the number of females and  $A$  the number of eggs in a gramme of faeces.



FIG 296 — HATCHING OF EGG OF *Ancylostoma duodenale* DUBINI 1843  
(From a photograph by J J Bell)

can be seen coiled up in the egg from which it escapes as a larva and feeds on the faecal material. The larva is needle shaped pointed posteriorly and measures 200 to 250  $\mu$  in length by 15 to 17  $\mu$  in breadth and is rhabdite in form with a long cylindrical terminal mouth opening into an oesophagus which after narrowing swells out into a bulb with three valves. The straight intestine surrounded by granular material opens into an anus situated some distance in front of the tip of the tail. This larva undergoes a first ecdysis when it becomes narrower and the oesophagus and mouth lose

months living on the food material enclosed in its own cells (stage of encystment). During this condition it may be quite active and can swim and climb up any surface which is wet. This is an important factor in explaining the production of certain kinds of skin eruptions. It is now ready to infect man which it does through the hair follicles of the skin causing eruptions or sores—e.g. ground itch.

From the hair follicles it forces its way via the

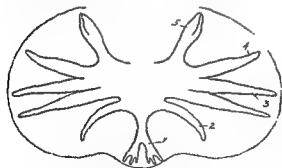


FIG. 203.—THE MEDIAN AND EXTERNAL CAUDAL BURSA OF *Ancylostoma duodenale* DUBINI

FIG. 204.—*Ancylostoma duodenale* DUBINI (After Looss)

1 Costa dorsalis 2 costa dorsalis externa 3 costa lateralis posterior et externa 4 costa lateralis med 5 costa ventralis

(After Railliet from Stiles's Report)

subcutaneous tissue into the venous bloodvessels and lymphatics. In the former it reaches the right heart and the lungs easily, in the



This history has been pieced together by Looss from observations on the infection of *Ancylostoma duodenale* in man and *A. caninum* in dogs.

These discoveries of Looss have been confirmed by Lambinet and Sambon while agreeing with the view that the worms penetrate the skin and work

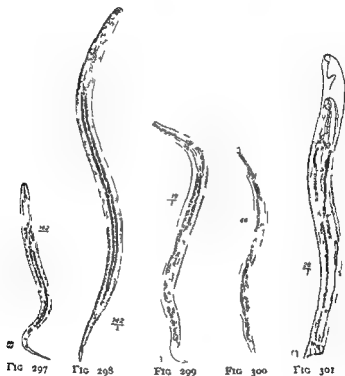


FIG 297      FIG 298      FIG 299      FIG 300      FIG 301  
 FIGS 297, 301—DEVELOPMENT OF *Ancylostoma duodenale* DEJEAN  
 (After Looss from Mensch)



*Trichinella spiralis* Owen, 1835

Synonym — *Trichina spiralis* Owen, 1835

*T. spiralis* then oh it occurred in the rat and afterwards in the pig

Sauces however are the greatest danger for in them the larvæ can live well protected

In order to infect man there must be a source of infection for the pig and this in the first instance can come from the rat and afterwards be kept up in the pig and then the transmission to man is easy

As rats pigs and men are cosmopolitan so trichiniasis is also cosmopolitan

tion They are mostly found in the diaphragm the larynx tongue abdo-

is formed

Pathogenicity.—When the female pierces the mucosa and during the wandering through the lymph and blood of the larvæ very severe symptoms called trichinosis or trichiniasis are produced

SUBFAMILY TRICHURINÆ Ransom, 1911

Type Genus — *Trichuris* also *Capillaria* Zeder 1800

*Trichuris* Roederer and Wagler 1761

Trichosomudæ with the anterior part of the body very long and thread like, and the posterior thicker portion sharply trun-

the bursa is bilobed. In the female the vulva lies in the anterior part of the body.

**Type Species**—*Necator americanus* Stiles 1902. Other species *N. africanus* Looss 1911 in the chimpanzee.

***Necator americanus* Stiles 1902**

**Synonyms**—*Dochmius duodenalis* R. Leuckart *pro parte* *Uncinaria americana* Stiles 1902 *Ancylostoma americanum* von Linstow 1903 *pro parte*

In 1902 Stiles discovered that two distinct genera were being confused under the term *Ancylostoma* and eventually called the new genus *Necator*. Leiper has recently shown that this new genus is very widely distributed over the world and is the common cause of ankylostomiasis in Ceylon while it occurs in India Assam



FIG. 302.—THE MOUTH CAPSULE OF *Necator americanus* STILES (After Looss from Mense)

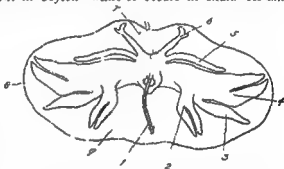


FIG. 303.—THE BURSA COPULATRIX OF A MALE *Necator americanus* STILES

1 Spicules 2 costa ventralis 3 costa lateralis posterior 4 costa lateralis externa et costa lateralis media 5 costa dorsalis externa 6 costa dorsalis 7 dorsal lobe 8 lateral lobe 9 ventral lobe

(After Stiles)

Burma, the Philippine Islands, Fiji, Japan, America, and has been found in West and Central Africa and in North West Rhodesia.

**Trichinella spiralis** Owen, 1835.Synon. = *Tr. spiralis* Owen, 1835.

Further, the larvae in the flesh of the pig are very difficult to kill and will resist a treatment which would destroy the larvae in other animals. Sausages protected.

In order to infect man there must be a source of infection for the pig.

present it is common also in America.

**Morphology and Life-History**—It is usually found in human or pig's muscles where it appears as minute white specks which when magnified

is formed

**Pathogenicity.**—When the female pierces the mucosa and during the wandering through the lymph and blood of the larvæ very severe symptoms called trichinosis or trichiniasis are produced.

## SUBFAMILY TRICHURINÆ Ransom, 1911

**Type Genus.**—*Trichuris*, also *Capillaria* Zeder 1800

**Trichuris** Roederer and Wagler, 1761

Species in man *Trichuris trichiura*  
Linnaeus 1761



FIG 304—*Trichuris trichiura* (LINNÆUS 1761) MALE  
(From a photograph by J J Bell)



FIG 305—*Trichuris trichiura* (LINNÆUS 1761) FEMALE

*Trichuris trichiura* LINNÆUS 1761

Synonyms—*Ascaris trichiura* L 1771 *Tri hoccephalus hominis*  
Schrank 1788 *T dispar* Rudolphi 1801

This worm is very commonly met with in the tropics. Its eggs appear in the feces and it is met with in the cæcum and sometimes



FIGS 306 AND 307—*Trichuris trichiura* LINNÆUS FEMALE AND MALE  
(After Claus)

the anterior  
while  
disease



TRICHURIS

cated, with a terminal anus Male with a spirally rolled pos

Linnaeus 1761



FIG 304 — *Trichuris trichiura* (LINNAEUS 1761) MALE

FIG 305 — *Trichuris trichiura* (LINNAEUS 1761) FEMALE

(From a photograph by J J Bell)

*Trichuris trichiura* LINNAEUS 1761

Synonyms — *Ascaris trichiura* L 1771 *Trichocephalus hominis* Schrank 1788 *T dispar* Rudolphi 1801

This worm is very commonly met with in the tropics. Its eggs appear in the faeces and it is met with in the caecum and sometimes



FIGS 306 AND 307 — *Trichuris trichiura* LINNAEUS FEMALE AND MALE (After Claus)

in the vermiform appendix during post mortem. Its anterior end is frequently found in the mucous membrane and

**Classification.**—The Marchantiales are closely allied to the families of the Thallophytes, and are distinguished from the other Thallophytes by the presence of a definite stem and leaves, and by the presence of a definite root system.

**DESCRIPTION**

Marchantiales are a distinct group of plants, and are distinguished from the other Thallophytes by the presence of a definite stem and leaves, and by the presence of a definite root system.

**Distichlis spicata (L.) Nees**

*Distichlis spicata* (L.) Nees

This species was found in the swamps of the Bay of Islands, and is distinguished from the other Thallophytes by the presence of a definite stem and leaves, and by the presence of a definite root system.

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A great many tropical countries appear to be plagued with leeches which affect men and animals as indicated above but it is probable that Algeria Palestine and Ceylon are the most infested. Among the other places in which they are troublesome may be mentioned the Philippines Java Sumatra Australia Japan and Chili. In Algeria and Palestine the leech lives in the pools of drinking water and here the endoparasitic form may be met with whereas in Ceylon it is usually a land leech which attacks the individual and therefore the ectoparasitic condition is common while the endoparasitic is more rare being due to *Hirudo multistriata*.

March 1 a 1 2

**Biology**—Leeches appear to be essentially water animals and though certain genera can live on land still they require a great deal of moisture. Hence land leeches retire into moist places under stones earth etc and only come out when requiring food. In dry weather not a leech can be seen while in damp weather they

— b. adant

animal approaches and set forth at once often with considerable speed to the attack.

The bite is not always painful at first and may not be noted until the flow of blood is observed. The leech while biting keeps itself and the skin of the victim moist by liquid excreted by the

affect them. Hence they when a human being or

**Rhabditis niellyi**

NIELLY (1883) Archiv Méd Nav xxvii 337 488

**Leptodera pelio**

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lxviii 248 251 Paris

Australia by Whitman in 1886 under the term *Geobdella* altered to *Moquimia* by Blanchard

<i>ceylanica</i> de Blainville 1827	Ceylon	<i>H. vagans</i> R. Blanchard	Madagascar
<i>umbata</i> Grube	Sydney	<i>H. javanica</i> Wahlberg	Java
<i>fallax</i> R. Blanchard	Madagascar	<i>H. talagalla</i> Meyen	Philippines
<i>morsitans</i> R. Blanchard		<i>H. japonica</i> Whitman	Japan

### Other Genera

ertures

### Remaining Orders

The remaining orders *Histriobdellida*, *Acanthobdellida* and *Branchiobdellida* do not concern us

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nephridia and the dermal glands. When it has gorged itself with blood it drops off but its salivary secretion prevents the coagulation of the blood and hence the site of the leech bite bleeds considerably for some time. The blood is stored up in the crop and its caecal diverticula and only a little is used day by day for food.

**Pathogenicity**—Leeches are hosts of trypanosomes and other parasites and their bite may possibly introduce these parasites

from the wound caused by the bite. Further the punctures caused by *Hæmadsysa zeylanica* are extremely liable to become ulcers which according to Marshall and Davy caused a high rate of mortality among the Madras sepoy and coolies during the Kandyan rebellion of 1818 in Ceylon. Short of death amputation of the limb was necessary in those days. We are not inclined to consider these old statements as erroneous because it is quite possible that some organism is often introduced into the affected part by the leech bite. For a further discussion of the pathogenicity see the chapters on Diseases of the Respiratory Organs and of the Skin.

to the body while the posterior is distinct

concerns us

#### FAMILY GNATHOBDELLIDÆ

*Arhynchobdella* ...

#### SUBFAMILY HIRUDININÆ R. Blanchard 1894

Aquatic Gnathobdellidæ with ten eyes and with an eyeless ring between the third and fourth pair of eyes with dentate jaws. Complete somites formed of five rings. The nephridial pores open near the margins of the body on the ventral surface.

**FAMILY POLYDESMIDÆ**—*Juliformia* with large free head and laterally extended dorsal plates—*Polydesmus complanatus* has once been recorded as an accidental parasite in the human alimentary canal in Europe

**Pathogenicity**—These parasites give rise to symptoms both direct and reflex

### CLASS III ARACHNIDA LAMARCK 1815

Euarthropoda with two pre oral segments the first bearing typical eyes and the second antennæ or chelicerae and six post-  
sessing  
chela  
nd the

higher pulmonary organs

The Arachnida are classified by Lankester into —

Grade A Anomomeristica

Grade B Nomomeristica

Subclass I Pantapoda

Subclass II Euarachnida

Grade a Hydropneusta

Grade b Aeropneusta

Section A Pectinifera

Order Scorpionidea

Section B Epectinata

Order I Pedipalpi

Order II Aranea

Order III Palpigrada

Order IV Solifuga

Order V Pseudoscorpions

Order VI Podogona

Order VII Ophiones

Order VIII Acarina

Order IX Linguatulida (Incertæ sedis)

Details with regard to this classification may be obtained by reference to Ray Lankester's paper in the *Quarterly Journal of Microscopical Science* 1904 vol xivm p 165

We have already considered the effects of the bites of the Scorpionidea the Aranea and some of the Acarina and now it is necessary to consider more particularly those which are parasitic and cause or spread disease

Two orders must be dealt with in some detail—viz : the Acarina and the Linguatulida

### ORDER ACARINA Nitzsch 1818

**Synonym**—Rhynchostomi

**Defini**

second

mouth

are wide ,

1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 32 33 34 35 36 37 38 39 40 41 42 43 44 45 46 47 48 49 50 51 52 53 54 55 56 57 58 59 60 61 62 63 64 65 66 67 68 69 70 71 72 73 74 75 76 77 78 79 80 81 82 83 84 85 86 87 88 89 90 91 92 93 94 95 96 97 98 99 100

## CHAPTER XXVIII

### ARTHROPODA

Arthropoda—Diplopoda—Acarina—Gamasoidea—Ixodoidea—Trombidoidea  
—Eupopoidea—Sarcoptoidea—Vermiformia—Linguatulida—Crustacea  
—Chilopoda—References

#### PHYLUM ARTHROPODA v Siebold and Stannius 1845

BILATERALLY symmetrical metazoa with well developed body cavity and heteronomously segmented body with hollow segmental appendages moved by intrinsic muscles and penetrated by blood spaces. One or more pairs of appendages behind the mouth are densely chitinized and turned inwards so as to act as jaws.

The phylum Arthropoda is divided into the following grades and classes by Ray Lankester —

#### GRADE A PROTARTHROPODA

Class I Onchophora

#### GRADE B EUARTHROPODA Lankester

Class II Diplopoda

Class III Arachnida

Class IV Crustacea

Class V Chilopoda

Class VI Hexapoda

The Onchophora which includes the genus *Peripatus* does not concern us but the other classes all contain species of importance in medicine.

#### CLASS II DIPLOPODA RAY LANKESTER 1904

Synonym.—Chilognatha

Euarthropoda in which the somites generally fuse after early development forming double somites with two pairs of appendages or two jointed legs and  
by trachea  
symphysis and

—Joints on each

broad dorsal

—two species of *Julus*—viz *J. terrestris* L. and *J. longipennis* Leach  
—have been found as occasional parasites (accidental) in the human alimentary canal in Europe

**Classification.**—The order Acarina is divided into the following suborders —

**SUBORDER NOTOSTIGMATA** —Acarina in which the opisthosoma anterior tracles

ment strengthened by chitinized dorsal and ventral sclerites integu Stigmata on the acetabula of the third fourth fifth and sixth pairs of appendages (Family Oribatidæ) This also does not concern us

**SUBORDER III METASTIGMATA** —Acarina with a hard integument like the Cryptostigmata One pair of stigmata above and behind the base of the fourth, fifth or sixth pair of appendages

Superfamily A Gamasoidea

Superfamily B Ixodoidea

**SUBORDER IV PROSTIGMATA** —Acarina with soft integument strengthened by special sclerites those on the ventral surface of sending the basal segments of the except in the aquatic species there above the first pair of appendages

Superfamily A Trombidoidea

Superfamily B Eupopoidea

**SUBORDER V ASTIGMATA** —Degenerate Acarina mostly parasitic, of integumental sclerites and the respiratory system absent

Degenerate parasitic Acarina without respiratory system and with the body produced posteriorly into an annulated caudal prolongation With the third, fourth fifth, and sixth pairs of appendages short and only three jointed

Acarina  
with pairs  
fifth and  
suborder,

however, need not concern us

### SUBORDER III METASTIGMATA

The parasites included in this suborder would in popular language all be called ticks

many

recurved teeth

### SUPERFAMILY A GAMASOIDEA

The superfamily Gamasoidea is divided into three families —(1) Dermanyssidae (2) Uropodidae (3) Gamasidae

distribution of the host—of the food—a remark the importance of which has already been appreciated. The observer will find that a parasitic disease is often bounded by the distribution of the food of the insect which spreads the real cause of the disease.

**Morphology**—The morphology is described under the heading Ixodoidea.

**Life-History**—The female lays eggs covered by a shell and an inner membrane called the deutovum. A six legged larva hatches out from the egg and after a time during which it may be active or quiescent moults and produces the nympha which is eight legged and resembles the adult except in the non development of the sexual organs and apertures. The leg which is added to the nympha is the fourth or posterior. The nymph is usually energetic and feeds on some host eventually passing into a quiescent stage. The male changes it moults and the males are usually smaller than the females. Both sexes suck fluid from animal or plant. Their life history

will be found to vary with the different families and species.

sucking their blood. Wellman has reported that he has caught *Phonergates bicoloripes* Stal in the act of sucking the juices of *Ornithodoros moubata* the tick responsible for the spread of Dutton's relapsing fever.



the tick fever of Africa Tsutsugamushi disease, the tick fever of the Rocky Mountains, etc They are parasitic on mammals birds

in tropical countries



FIG 310—*Margaropus annulatus*  
VAR *australis* SAY DORSAL VIEW  
OF THE MALE

ultimate penultimate and apical

palpi

3 The hypostome is an elongated structure composed of two symmetrical halves with numerous minute teeth

in the body is swollen for the attachment

teeth

5 The mandibular sheath lies dorsal to the mandibles

6 The palpi are composed of four segments or articles—basal antepen They are of importance in classification

1 Dorsal Surface—On the dorsal surface may be noted (Figs 313 and 318) —

(a) The scutum found in the *Ixodidae* and well marked in the adult male but much smaller in the adult female It is a hard chitin

(d) The postero-marginal festoons when present are eleven

## FAMILY I DERMANYSSIDÆ

Gamasoidea parasitic on vertebrates with mandibles fitted for piercing with the body sometimes constricted with soft integuments finely striated

The Dermanyssidæ are divided into two subfamilies — Dermanyssinæ with an anal plate Halirachinæ without an anal plate

## SUBFAMILY DERMANYSSINÆ

*Dermanyssus* Dugès 1834

Dermanyssinæ with a long body not distinctly constricted peritreme on the venter with chelate mandibles in the male and long stiliform mandibles in the female Parasitic on birds

*Dermanyssus gallinæ* Recl. 1874

metre

*Holothyrus* Gervais 1842

*Holothyrus coccinella* Gervais 1842

## FAMILY 2 GAMASIDÆ

## SUBFAMILY II IXODOIDÆ BANKS 1894

Synonyms — *Acarus* Linneus 1746 *pro parte* *Ricinia* Latreille 1804 *pro parte* *Ixodides* Leach 1815 *Ixodea* Burmeister 1837,

The  
these arachnids as *κυσσαυρις* (the dog tormentors) because ticks were well known in his day to attack hunting dogs

The Ixodoidea are the ticks which have become of importance in tropical medicine not merely because of their unpleasant bite but because of the spread of disease by their agency — as for example,

The legs are segmented into the following articles: coxa, trochanter, femur, patella, tibia, and tarsus.

**Internal Anatomy**—The internal anatomy has been studied by Christy and Bonnet in eight

which lies the epidermis. A large cephalic gland is at the junction of the rostrum

with the body.

The mouth parts are the

maxilla

and

labrum

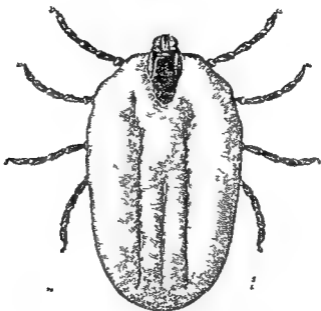


FIG 313 — *Vargarepus annulatus* var. *australis* SAY DISTENDED FEMALE

marked out by grooves lying along the posterior margin of the

(a)

(f)

2 *Ventral Surface*—The ventral surface exhibits (Fig. 314) —

(a)  $\pi$ 

(b)

(c)

in the male

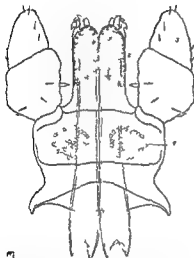


FIG. 311—DORSAL VIEW OF THE CAPITULUM OF A TICK FEMALE (*Haemaphysalis punctata* C AND F)

1 Chelicerae; 2 hypostome; 3 palps; 4 porose area

(After Nuttall Cooper and Robinson *Journal of Parasitology*)



FIG. 312—CHELICERA OF *H. punctata* MALE (C AND F)

(d) The stigmata lie one on each side of the body between the 3rd and 4th pairs of legs in the Argasidae and behind the fourth in the Ixodidae

(e) There are often to be seen a pair of genital furrows and an anal furrow

(f) Pits pores hairs and punctations are to be found as on the dorsal surface

3 *Anterior Margin*—This varies as compared with the posterior being sometimes straighter sometimes emarginate and receiving at the anterior end the rostrum

4 *Posterior Margin*—This is generally rounded and often marked by the festoons already mentioned on the dorsal surface

5 *Lateral Margins*—They vary as to their straightness or degree of curvature

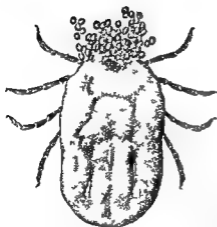


FIG. 115.—A TICK LAYING EGGS  
(After Sambon)

consists of a median heart and distributing vessels. The fat body is well marked.

There are a number of dermal glands, a coxal gland in the second coxal joints and opening on the first coxal joints by a minute pore and a cephalic gland in the head.

**Life-History**—While on the host sucking blood the male and female parasites copulate and the latter growing to a large size drops to the ground and lays a number of eggs. The egg consists of a shell with an inner membrane enclosing food yolk and embryo which eventually hatches as a six-legged larva without sexual organs or stigmata. The

or females with fully developed generative organs

The adults now become parasitic and moult and the young female fixing itself to the host grows considerably but rarely changes her place while the male remaining small wanders about looking for the female.

The life history and the habits of the different divisions of the *Ixodidae* are so various that they will be described under their separate heads. One example may however be mentioned here

which has been studied off the host temperature variation of the

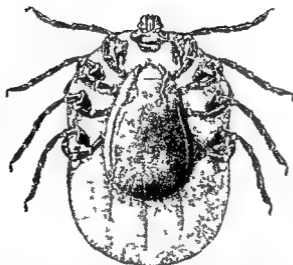


FIG 374 — COPULATION OF THE MALE AND FEMALE TICK  
(After Sambon)

lateral diverticulum subdivided into three branches—a medio-lateral into two or three a postero-lateral which is single and a posterior set which is also single

The walls of the central tube and the diverticula consist of a single layer of large cells lying upon a thin basement membrane.

thirty first day Thus in this case the larva and nymph remain parasitic on the host and do not drop off as described above

**Parasite on Man**—The ticks known to be parasitic on man are—(1) *Argas reflexus* Fabricius (2) *A persicus* Oken (3) *Ornithodoros savignyi* Audouin (4) *O moubata* Murray (5) *O megnini* Duges (6) *O turicata* Duges (7) *O tholozani* Laboulbène and Mégnin (8) *Alectorobius talaje* Guerin Méne

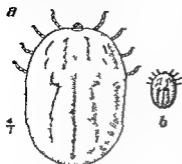


FIG 319—FULLY REPLETE FEMALE OF *Hamaphysalis pusillata* C AND F

(After Nuttall Cooper and Robinson *Journal of Parasitology*)

(a) Magnified (b) natural size

(9) *Dermacentor reticulatus* Fabricius (10) *D andersoni* Stiles (11) *D electus* Koch (12) *D modestus* Banks (13) *Ixodes ricinus* Linnæus (14) *I hexagonus* Leach (15) *Amblyomma americanum* Koch (16) *A dissimile* Koch (17) *Hyalomma ægyptium* Linnæus.

**Pathogenicity**—Ticks are spreaders of disease in man and animals

They may be classified into spreaders of *Spirochætidæ* spreaders of *Babesia* and into spreaders of unknown germs

### 1 Spreaders of Spirochætidæ

1 *Ornithodoros moubata* carries *Spiroschaudinna duttoni* Novy and Knapp 1906 and causes African tick fever or Dutton's relapsing fever in man

2 *Argas persicus* carries *Spiroschaudinna marchouxii* Nuttall 1904

3 *Margaropus annulatus* carries *Spiroschaudinna theileri* Laveran 1904

### 2 Spreaders of Piroplasma

### 3 Spreaders of Unknown Germs

1 *Argas persicus* causes a disease in Persia 2 *Dermacentor tenellus* (see *D andersoni*) causes Rocky Mountain fever Experimentally the nymphs of *D marginatus* and *Amblyomma*

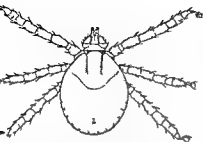


FIG 316—THE LARVA OF *Hamaphysalis punctata* C AND F (X 40)  
After Nuttall Cooper and Robinson (*Journal of Parasitology*)

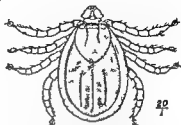


FIG 317—THE NYMPH OF *Hamaphysalis punctata* C AND F  
(After Nuttall Cooper and Robinson (*Journal of Parasitology*))

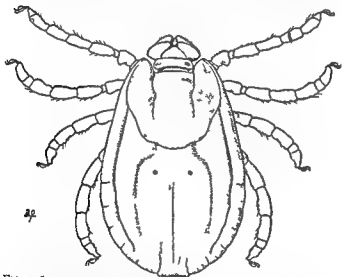


FIG 318—ADULT FEMALE OF *Hamaphysalis punctata* C AND F  
(After Nuttall Cooper and Robinson (*Journal of Parasitology*))

...uch they moult and form nymphs on the sixth day of their para  
ic existence—; e without leaving the host The nymph sucks



by several males. The female now becomes quiescent, and passes out to give a process of development, which is completed

generally laid in a loosely adherent mass

The six-legged larva hatches in less than a week. In *Ornitho-*

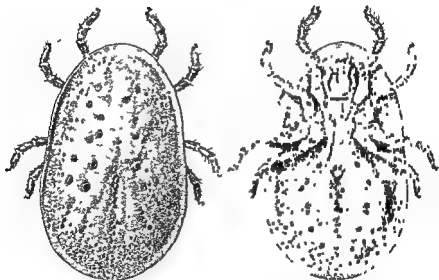


FIG 320 — *Argas persicus* LATREILLE  
FEMALE DORSAL ASPECT

FIG 321 — *Argas persicus* LATREILLE  
FEMALE VENTRAL ASPECT

The adult Argasidæ appear to moult many times as they grow

four genera — *Argas*,

1. Eyes absent, body usually flat, with thin borders, without  
— *Argas*, (b) trans

s distended, with  
deep ventral grooves (a) Without a fold of SKIN (sclerite) on each  
side of palpi — *Ornithodoros*; (b) with such a fold — *Alectorobius*

*Americanus* and the nymphs and adults of *D. variabilis* can transmit the virus of Rocky Mountain fever

Example The —

Powder) It was found to be

is very useful

**Quarantine**—Quarantine of cattle and control over their movements is important

There are however

**Classification**—The Ixodoidea are divided into two families —  
**FAMILY 1 ARGASIDÆ** Canestrini 1890—Ixodoidea without a scutum mouth parts of adult not prominent from above, no pulvillus attached to tarsus in adults

**FAMILY 2 IXODIDA** Murray 1877—Ixodoidea with a scutum mouth parts prominent from above pulvillus present attached to the tarsus in adults

Example

## FAMILY 1 ARGASIDÆ

There are

the fluid between the parasite and the skin of the host is alkaline and prevents coagulation of blood After feeding the tick from the host

becomes active again and is ready for another feed

The Argasidæ pass at various times one female being fertilized

This *Argas* is widely distributed being found in Asia where

tick of the United States and the adobe tick of Mexico and Arizona. According to Balfour, it spreads the *Spirochæta marchouxi* in Sudanese fowls. Nuttall and Strickland have demonstrated the presence of an anticoagulum in the salivary glands and intestine of the tick.

*Argas brumpti* Neumann 1907

This tick was discovered by Brumpt in Somaliland. The dorsum is marked by symmetrical hexagonal depressed areas. Female 20 by 13 millimetres. Nuttall says that according to Brumpt the bite is very painful and causes pruritus lasting several days and the site may remain indurated after seven years.

*Argas cucumerinus* Neumann 1901

Only the male of this tick is known. It has an oval elongated body brownish red in colour 10 by 5 millimetres. It is found in Lima in Peru. Possibly this is merely a variety of *A. reflexus*.

*Argas hermanni* Audouin 1827

*Argas* with very fine skin folds and small rostrum. It is found in Abyssinia and Egypt. Possibly this also is only a variety of *A. reflexus*.

*Argas æqualis* Neumann 1908

It was found in  
5 by 2.5 milli

*Argas transgariëpinus* White 1846

Synonym — *Argas kochi* Neumann 1901

*Argas* with very compressed body not much longer than broad — 7.5 by 6 millimetres. Margin with irregular folds. Found in South Africa.

*Caris* Latreille 1804

*Argasidæ* with almost circular body a little larger in front than behind with a conspicuous transverse groove behind the anus.

*Caris vesperthionis* Latreille, 1796

Synonyms — *Caris vesperthionis* Latreille 1796 *Argas pulchella* George 1876  
Parasitic on bats

There is, however, some doubt as to whether *Caris* is really entitled to be a separate genus

*Argas* Latreille, 1796

Synonyms.—*Nec Argas* Scoul., 1835, *Caris* Latreille, 1796,

around the base of the rostrum ventrally tegument roughened by wrinkles and folds, and marked by circular pits Without eyes

Type.—*Argas reflexus* Fabricius, 1794

The number of species known is steadily increasing The more important can be defined by the following table, modified from Neumann —

A Posterior margin with rectangular festoons—*A persicus*

B Posterior margin with narrow festoons —

I. Tegument with fine folds, body long, swollen—*A hermanni*

II Tegument with large folds, body flat —

(a) Body narrower in front —

(1) Anterior extremity flat and rounded—*A reflexus*

(2) Anterior extremity pointed —

(a) With deep camerostome and dorsal hexagonal markings—*A brumpti*

(b) Without these—*A equalis*

(β) Body almost as large in front as behind —

(1) Body short, a little longer than broad—*A transgarpinus*

(2) Body twice as long as broad—*A cucumerinus*.

*Argas reflexus* Fabricius, 1794

Synonyms.—*Acarus reflexus* Fabricius, 1794, *A marginatus* Fabricius, 1794, *Rhynchoporon columbae* Hermann, 1804

*Argas* with yellowish body, male 4 by 3 millimetres, female 6 to 8 by 4 millimetres Its distribution is mainly in Europe and America, where it lives in dovecots but it can and does attack man, producing the symptoms already described

*Argas persicus* Oken 1818

Synonyms.—*Rhynchoporon persicum* Oken 1818, *Argas persicus* Lischer de Waldheim, 1820, *A mauritanus* Guérin Méneville, *A minutus* Koch, 1844; *A americanus* Packard 1872, *A sarchezi* Dugès, 1891; *A chinche* Gondet

*Argas* with oval, brownish red body Male 4 to 5 by 3 millimetres, female, 7 to 10 by 5 to 6 millimetres Dorsal and ventral surfaces with pits in rows, and irregularly placed

It is common in Africa, India, and Aden, and perhaps elsewhere

second and third pairs. Body oval constricted slightly between the third and fourth pairs of legs, yellow in colour when young and blackish brown when old. Integument covered with irregular hemispherical prominences pointed at their summits between which are narrow depressions with hairs. Capitulum embedded in an infundibulum.

without middle apophysis. Teeth of the hypostome arranged in two rows. Two spines at the base of the

two transverse furrows one just in front of the posterior border and the other situate farther forward marking off a median elevation with a central depression and seven similar depressions each of which becomes a deep sulcus posteriorly

all marked are anal furrow which is round the anterior defining the supra behind the anus are three symmetrical longitudinal grooves with a short V shaped depression abuts on the anus. The genital papilla is well marked

times as long as the first in size from the first to ung of the coxal gland. Christophers has observed that the fluid from this gland is alkaline and prevents coagulation of the blood. The stigmata are placed behind and above the supracoxal folds. The fifth joints of the first three pairs of legs with three teeth the two proximal quadrangular and the distal conical

This tick is very hardy and is said to be able to live for many months without food or moisture. It attacks human beings and is found in Africa and India.

Pathogenicity.—Not known

*Ornithodoros moubata* Murray 1877

held Cobbold

Uganda, moubata in Angola, and Christophers has described a similar tick in Brazil

## Ornithodoros Koch 1844

Argasidae with or without eyes. Rostrum surrounded ventrally by a camerostome. Tips of the palpi visible from above. Lateral borders of the body straight, sometimes concave. Integument mammillated with hemispherical elevations. Two longitudinal coxal folds, a pair of supracoxal folds, one transverse pre-anal and one post-anal groove, and one longitudinal anal groove running from the anus to the anterior end.

## DIAGNOSTIC TABLE

## A Eyes present —

*O. pavementosus*

## B Eyes absent —

- I Subrectangular body nearly square (Mexico)—*O. turiscatus*
- II Body more or less constricted behind the fourth pair of legs —
  - (a) With broad anterior end (Mexico)—*O. megnini*
  - (b) With narrow anterior end (Lahore)—*O. lahorensis*
- III Ovoid body with broader posterior end —
  - (a) Anterior end broad and rounded (Central Africa)—*O. monbata*
  - (b) Anterior end tapering —
    - (1) Tarsi forked distally (Ecuador)—*O. furcosus*
    - (2) Tarsi slightly knobbed (Algeria)—*O. erraticus*
    - (3) Tarsi second to fourth markedly knobbed (Persia)—*O. tholozani*
    - (4) Tarsi first with three distal knobs, second to fourth with one knob (Persia)—*O. canestrinii*

## Ornithodoros savignyi Audouin 1837

Synonyms — *Argas savignyi* Audouin 1827, *Ornithodoros mortbilosus* Gerstäcker 1873, *Argas schineri* Berles 1883.

This is the type species of the genus, and has been studied in detail by Neumann and Christophers.

## ARTHROPODA

• • *dors* *turcatus* Duges 1876

*ricata* Duges 1876 *Ornithodoros americanus*

Marx 1895

*Ornithodoros* without eyes anteriorly the body is much narrowed  
Tibiae and tarsi with three small tubercles

3 **Habitat**—Mexico and Central America Attacks men and  
fowls

***Ornithodoros megnini* Duges 1883**

18

Females 5 to 6 millimetres by 3 to 4 millimetres Males somewhat  
smaller

This is the ear tick of American cattle and has been found in  
the ear of man

***Ornithodoros lahorensis* Neumann 1908**

*Ornithodoros* without eyes Male 8 by 4.5 millimetres Female  
10 by 5.6 millimetres Found at Lahore India Parasitic on sheep

***Ornithodoros tholozani* Laboulbene and Megnin 1882**

**Synonyms**—*A tholozani* Laboulbene and Megnin 1882 *A papillipes* Birula 1895

*Ornithodoros* without eyes Males 4 to 6 millimetres in length  
and 2 to 4 millimetres in breadth Females 8 to 10 millimetres by  
4 to 5 millimetres

It is specially a parasite of sheep in Caucasia and Persia but is  
very dangerous to man

***Ornithodoros pavementosus* Neumann 1901**

*Ornithodoros* with eyes Body covered with flat warts forming  
a pavement South Africa Only female known 12 by 8 millim-  
etres

***Ornithodoros fuscus* Neumann 1908**

Found in Ecuador Female 10 by 5 millimetres

***Ornithodoros erraticus* Lucas 1849**

**Synonyms**.—*Argas erraticus* Lucas 1849 *O miharis* Karsch 1860  
Found in Algeria and Bengal Length 5 millimetres breadth  
3 millimetres

***Ornithodoros coriaceus* Koch 1844**

*Ornithodoros* with eyes Only one knob on the hind tarsus  
America Male 6.4 to 8.6 by 3.4 to 4.6 millimetres female 9.5 to  
13.8 by 5.3 to 8.2 millimetres

**Morphology**—*Ornithodoros* without eyes body oval a little wider behind than in front with a slight constriction between the third and fourth legs colour varies with age from yellow brown to dusky brown Integument

ars of pits from each of which a sulcus runs back wards and inward.

The ventral surface shows a well marked pre-anal sulcus which joins the supracoxal groove as in *S. savignyi* behind the anus are three pairs of longitudinal grooves The stigmata are semilunar situated above the supra-oxal groove The last segment of the fourth pair of legs is stout and con-

with three teeth—proximal

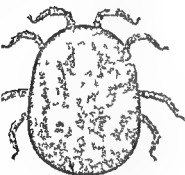


FIG 322—*Ornithodoros moubati* MURRAY FEMALE DORSAL ASPECT (X 4)



FIG 323—*Ornithodoros moubati* MURRAY FEMALE VENTRAL ASPECT (X 4)

**Life-Hist** —

70 to 139

891 by 77

becoming

temperature of 20° to 30° C and a humidity of 71 to 77 per cent After three or four days these nymphs suck blood At first the position of the stigma is marked by only a small white spot and not but after the — —

blood and may live rather resemble bugs fluid secreted by the

coxal glands prevents coagulation of the blood

**Pathogenicity**—It is the spreader of *Spiroschaetium dilorzi* and according to Wellman and Feldmann possibly of *Acarthocheilus neva perstans*



shivel and to show yellow areas due to the distension of the Malpighian tubes with guanine. At the end of oviposition the tick dies.

From the egg comes a six legged larva which generally climbs into some grass or bushes and waits to get on to a host. While so doing it may remain for months without food.

It now sucks blood and drops off this first host and moults on the ground becoming an eight legged nymph like an adult but without generative apparatus which again has to go in search of a second host and obtain a feed of blood when it drops off and undergoes a second moult turning this time into the sexually mature adult which goes in search of a third host. Before feeding the adult female is small flat and thin. When she arrives on the third host she drives her rostrum perpendicularly into the skin as far as its base a hole being made by the mandibles.

The palpi do not enter the hole in the skin but lie on each side.

The recurved hooks on the hypostome keep the tick in position and can only with difficulty be detached. The tick now sucks the blood and at the same time becomes fertilized and increases enormously in size by the addition of blood and the development of the eggs. She then drops off this third host and proceeds to lay her eggs.

ich in size

#### SUBFAMILY 1 RHIPICEPHALINÆ Salmon and Stiles 1901

Synonyms — *Rhipistomidea* Koch 1844 *Conipalpi* Canestrini 1890 *Rhipicephala* Neumann 1897 *Rhipistomida* Marx 1896  
*Ixodida* in which the palpi are no longer than broad. Anterior

*ephyialis*; *Dermia*

#### SUBFAMILY 2 IXODINÆ Salmon and Stiles 1901

Synonyms — *Ixodidea* Koch 1844 *Cultripalpi* Canestrini 1890  
*Ixodida* Marx 1892 *Hematostorida* Marx 1892 *Eschatocephalida*

broad rostrum long Anterior  
irginate

Type Genus — *Ixodes*

Genera — *Ixodes* *Eschatocephalus* *Ceratixodes* *Aponomma*  
*Amblyomma* *Hyalomma*

**Ornithodoros canestrinii** Birula 1895

Synonym — *Ixas canestrinii* Birula 1895

Found in Persia Male 10 by 5 millimetres female 14 by 5 millimetres

**Alectorobius** Pocock 1907

Argasid ♀ with folds of integument capable of being folded under the palpi This genus is not recognized by Nuttall as he considers it a synonym of *Ornithodoros*

Type — *A. talaje* Guérin Méneville 1849

**Alectorobius talaje** Guérin Méneville 1849

Synonyms — *O. talaje* Guérin and Méneville 1849 *O. ruidis* Harsch 1880 *Alectorobius talaje* Pocock 1907

This is the chinch of South America and Mexico where it is a great pest *A. coniceps* of South Europe and *A. capensis* of South Africa are varieties of this species

## FAMILY 2 IXODIDÆ Murray 1877

Synonyms — *Ixodes* Duges 1834 *Ixoides* Koch 1844 *Ixoides* Gervais and van Beneden 1859 *Ixolius* Canestrini and Fanzago 1877 *Ixoline* Trouessart 1892 *ixistomata* Marx 1892

Ixodoidea with a dorsal scutum and a terminal capitulum The digit of the mandible has two apophyses and the palpi are free

Most of the ticks belong to this family which has the following features —

1. . . . .

shortest and the fourth pair the longest The tarsus has a pulvillum The stigmata are situate posterior to the coxa of the fourth leg The male is smaller and fatter than the female The scutum covers the whole dorsum except a marginal region the posterior portion of which between the two stigmata is generally divided into eleven festoons The female has but a small scutum situated anteriorly and the capitulum has two symmetrical porose areas on its basal piece

Type Gen . . . . .

Life-Hl . . . . .

drops off

by its legs

the interior part of the ventral surface between the first pair of legs becomes depressed and forms a hollow in which the head and genital orifice are situated When an ovum presses out of the orifice the tick moves slightly backwards This oviposition takes about fifteen to twenty days While it is proceeding the tick begins to

small tick

the place to

the and the

at each side Palpi short and broad : First coxa with two large teeth Male with one or two pairs of anal shield (Fig 374)

Type Species —*Eurhipicephalus sanguineus* Latreille 1804

### *Eurhipicephalus appendiculatus* Neumann 1901

This is the brown tick of South Africa where it spreads *Theileria parva* among cattle and buffaloes causing coast fever

Morphology —Male scutum does not quite cover the dorsum festoons narrow the median is prolonged into a caudal process In front of the festoons three wide longitudinal grooves Size 4 by 2.6 millimetres Female dorsal plate oval porose area small

Life-History —The eggs are laid in thousands on the grass and hatch in about twenty eight days into six legged larvæ which pass from the grass on to cattle suck blood and drop off replete in about three to four days.

They now remain dormant for about twenty one days and finally

ing the  
te and  
t m la

What becomes of the male is not known

Pathogenicity —It spreads *Theileria parva* the cause of coast fever in cattle by the bites of the infected nymphs only—the infected larvæ and adults do not spread the disease but the larva can hand the infection over to the nymph which is the spreader

### *Eurhipicephalus bursa* Canestrini and Ganzago 1878

also  
ttle

and dog tick

Morphology —Male scutum half as long as wide narrow anteriorly with many punctations Eleven festoons Size 4.5 by 3 millimetres

Female when newly hatched flat when distended with blood  
b. at the middle of the length Tegula

velop  
eight

days forming nymphæ which grow for twenty one days fall off on to the ground moult and become adults which again infest sheep

## DIAGNOSTIC TABLE OF GENERA FROM SALMON AND STILFS

## RHIPICEPHALINÆ

## A Eyes present —

I Dorsal surface of capitulum hexagonal sides drawn out laterally into sharp points

## (a) Males with anal plates

1 Second and third palpal segments straight, stigmata comma shaped—*Eurhipicephalus*

2 Second and third palpal segments drawn out laterally into sharp points stigmata round—*Margaropus*

(b) Males with rudimentary anal plates—*Rhipicestor*

II Dorsal surface of capitulum rectangular sides straight, male without anal plate—*Dermacentor*

B Eyes absent—*Hæmaphysalis*

Nuttall and Warburton's new classification of the Ixodidae is as follows —  
*Prostrata*—Ixodidae with anal grooves surrounding the anus in front—

*Ixodes*

*Metastriata*—Ixodidae with anal groove contouring the anus behind but this groove may be faint or obsolete

*Brevirostrata* —

Group I Inornate without eyes but with festoons—*Hæmaphysalis*

Group II Ornate or inornate with eyes and with or without festoons

Anal grooves marked

Ornate with festoons

Basis capituli rectangular dorsally—*Dermacentor*

*Longirostrata* —

Group I Ornate or inornate with eyes and with or without festoons

## C

developed or no eyes

It will be observed that the genera *Eschatotephalus* and *Ceratixodes* are rejected and referred to the genus *Ixodes*. Another genus *Neumannella* Lahille 1905 is also rejected and it is referred to *Aponomma*.

## SUBFAMILY RHIPICEPHALINÆ

*Eurhipicephalus* Neumann 1904

Synonyms — *Rhipicephalus* Koch 1844, *Phaulosixodes* Berlese, 1880

Rhipicephalinæ with distinct eyes base of the capitulum broader than long hexagonal or dorsal surface forming a projecting angle

The male is 3.35 by 1.55 millimetres, with a scutum covering the dorsal surface, except at the sides and back. External apophysis with only two teeth.

*Eurhipicephalus evertsi* Neumann, 1897.

This is the red leg tick, which spreads *Nuttallia equi*, and is found in Europe, Africa, and Asia. The life-history resembles *Eurhipicephalus appendiculatus* in the changes of host by larva and nymph. The infection is acquired in the nymphal stage and transmitted by the adult. Size male, 5 to 6 by 3 to 4 millimetres, female, 14 by 9 millimetres.

*Margaropus* Karch, 1879

**Synonyms.**—*Boophilus* Curtice, 1891, *Rhipicephalus* Neumann, 1897.

*Rhipicephalinae* with eyes, though often indistinct; base and capitulum broader than long. Palpi short and broad, second and third segment thicker in the middle, and forming a sharp angle externally. Posterior margin of the first coxa slightly bidentate; stigmal plate round. Body without marginal festoons, anal plates

no species, of which two  
be one, the third being

*Margaropus annulatus* Say, 1821.

**Synonyms.**—*Ixodes annulatus* Say, 1821, *Hæmaphysalis rosea*

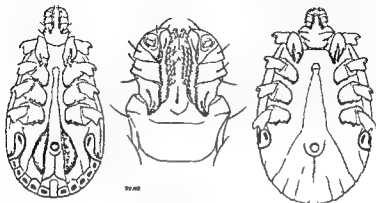
small sexual aperture and sexual furrows. Stigmata oval. Capitulum very short. Internal apophysis of mandible conical, the external with three teeth. Hypostome broad. Palpi very short. Legs short (*vide* Fig. 313).

Male with body oval, 2.15 by 2.05 millimetres, widest opposite stigmata. Scutum dorsal surface with large puncta in front of the level of the second pair of legs. Festoons hardly

Internal apophysis of the mandible with two teeth. Coxa of mandible directed posteriorly.

but not being found in Japan, and hence the species receive a little change in the name.

**Pathogenicity**—It spreads *Pyroplasma ovis*, which causes 'heart from an adult female ext generation which



FIGS 324-326 —*Eurhipicephalus pulchellus* GERSTACKER 1873  
VENTRAL ASPECT

a Male (X 10) b mouth parts (more highly magnified) c female (X 10)

#### *Eurhipicephalus simus* Koch 1844

**Synonyms**—*Rhipicephalus senegalensis* Koch 1844 *Rh. frater tatus* Gerstäcker 1893

This tick is found in various parts of Africa—viz Egypt, late German East Africa and the Cape. It has several varieties—e.g. *E. simus erlangeri*, *E. simus hilgerti*, *E. simus simplex*. In South Africa it is called the black pitted tick because of its punctations and causes coast fever by spreading *Theileria parva*. Male oval rounded posteriorly 4 by 2.5 millimetres. Scutum brown red. Female oval 6 by 3 millimetres.

#### *Eurhipicephalus sanguineus* Latreille 1804

wl

in front than behind  
Scutum very small  
integument nearly or completely without hairs. The mandibles have an internal apophysis with three teeth arranged one internally and two externally and an external apophysis with three teeth arranged in series.

coxa of the fourth leg much larger than those of the others Scutum ornamented

**Type.**—*Dermacentor reticulatus* Fabricius 1794.

In 1910 Stiles classified the species of *Dermacentor* into four groups according to the microscopical structure of the stigmatal plates in the adult

- A Adults with four longitudinal rows of large denticles on each half of hypostome, stigmatal plate nearly circular without dorso-lateral prolongation goblets very large—*D nitens*  
 B Adults with three longitudinal rows of large denticles on each half of

Say, 1821

**Remarks.**—There has been the greatest confusion as to the tick

have since been shown by him to be quite distinct. Therefore in any reference to a tick causing Rocky Mountain fever, no matter what name is used, it is important to understand that *D. andersoni* Stiles, 1905, is the species really referred to

***Dermacentor reticulatus* Fabricius 1794**

*reticulatus*  
*bicollis*  
1844.

***Dermacentor occidentalis* Marx 1892**

**Life-History** — *Margaropus annulatus* begins its life with the eggs on the ground from which the larva emerges and gaining access to

host and lays her eggs

**Pathogenicity** — It is the spreader of *Piroplasma bigeatum* the cause of Texas or red water fever in cattle. The adult female acquires the *Piroplasma* and passes it on to the larva which infects the

188

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Afr

dat

in Buenos Ayres

#### *Margaropus decoloratus* Koch 1844

This is looked upon as a variety of *Margaropus annulatus* Say by some authors. It is the blue tick of South Africa.

**Morphology** — The clypeal plates in the male end in sharp points and a caudal appendage is present. The hypostome has six rows of teeth.

**Life-History** — It lives from the larval stage to the adult on the same host. The adult when fully fed drops off and lays the eggs on the ground.

**Pathogenicity** — It is a spreader of *Piroplasma bigeatum*.

#### *Rhipicephor* Nuttall and Warburton 1908

1910

Basis capituli  
cranial angles with  
male resembles

... of ... and *Dermaacentor* ... north. Cox IV is  
piracles

#### *Rhipicephor* Meerns Nuttall and Warburton 1908

Synonym ...

1908

... in the Congo and  
Another species is

C  
I

#### *Dermaacentor* Koch 1844

*Rhipicephal* with eyes base of capitulum rectangular broader than long. Dorsal median porose plate present. Palpi short and thick. Stigmata comma shaped. Male without anal shields. The



mounts	The nymph attacks another dog feeds drops off and
moults	The adult attacks a third dog feeds drops off and lays

## SUBFAMILY IXODINÆ

The following diagnostic table modified slightly from Salmon and Stiles will indicate the genera —

A Eyes absent —

I F —

II Post anal crescentic groove open anteriorly—*Aphonomma* (Fig 333)

III Without anal groove in the female one anal shield in the male—*Ceratixodes*

B Eyes present —

I Anal plates absent—*Amblyomma* (Fig 336)

II Anal plates present on males—*Haalomma* (Fig 339)

*Ixodes* Latreille 1796

scutum posteriorly

Male with scutum not covering the lateral and posterior margins no festoons stigmata oval Ventral shields seven in number one pregenital one median two epimeral one anal and the adanal

Female with three dorsal longitudinal grooves and two longitudinal genital grooves ventral and the anal crescentic groove already mentioned

Type Species —*Ixodes ricinus* Linnæus 1758

*Ixodes ricinus* Linnæus 1758

Synonyms —*Acarus redurnus* Linnæus 1758 *A ricinus* Linnæus 1758

This is the castor oil tick and is found in Europe North Africa and North America on man sheep goats cattle horses dogs cats rabbits bats birds etc

genital grooves close together at first but diverging laterally behind the fourth coxa and ending between the second and third external festoons. Anus with short anomarginal groove. Stigma comma-shaped.

Capitulum with the posterior lateral angle prolonged into a sharp point. Porose areas circular. Internal apophysis of the mandible with strong posterior tooth external with three successive teeth. First coxa bidentate, other three with spines. Replete female. Swollen body laterally constricted at the stigmata of deep brown or slate colour. size 16 by 10 millimetres.

**Life-History**—The egg

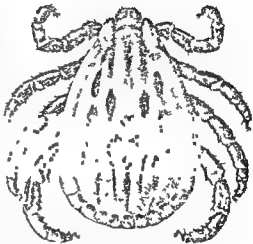


FIG 327—*Dermacentor andersoni* STILES 1909  
MALE DORSAL ASPECT

a which feeds on some

#### *Dermacentor andersoni* Stiles 1905

**Synonyms**—*Dermacentor occidentalis* of all writings on Rocky Mountain spotted fever until some time after 1910, *D. venustus*

or quite circular.  
Eyes not prominent  
or small punctations.

gated transversely Mandibles with two teeth on the internal apophysis and external with five teeth

**Life-History**—The female lays about 1000 eggs in about one to two weeks the eggs take six weeks to hatch into a larva which remain one week on the first host and then four weeks on the earth before it becomes a nymph This stage requires one week on the second host and eight weeks on the earth before it becomes the adult which seeks the third host copulates sucks blood and drops off to lay eggs

**Pathogenicity**—It acquires *Piroplasma bigeminum* from infected cattle in the adult stage and spreads it to fresh cattle in the larval and nymphal stages

*Ixodes hexagonus* Leach 1815

**Synonyms**—*Ixodes autumnalis* Leach 1815 *I. erinacei* Audouin Koch 1847

I tick can transmit  
*Piroplasma canis* Piana and Galli Valerio 1895

*Eschatocephalus* Frauenfeld 1853

**Synonyms**—*Sarcomissus* Kolenati 1857 *Hæmalastor* Neumann 1889

Ixodinae without eyes and with a long rostrum Palpi pyriform in the male and claviform in the female Pre anal groove opening posteriorly Stigmata circular Legs long

Male with dorsal and ventral irregular chitinous thickenings.

Female with very fine parallel grooves

**Type Species**—*E. vesperthionis* C. L. Koch 1844

There are over seven species found on bats and in caves

*Aponomma* Neumann 1899

**Synonym**—*Ophisodes* Murray 1877

Ixodinae without eyes and with the base of capitulum usually pentagonal with dorso lateral border very short palpi long Post anal groove Ventral sexual grooves

Male nearly as broad as long with a scutum marked with green spots covering the whole dorsal surface

Female scutum shorter than broad

**Type Species**—*Aponomma gervaisi* Lucas 1847

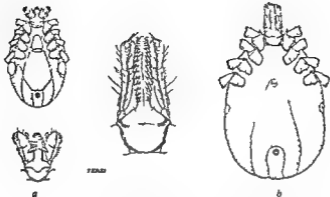
These ticks are found chiefly on reptiles but are also found on other animals

*Ceratixodes* Neumann 1904

Ixodinae with long palpi without eyes and without anal groove in female Stigmata circular One anal and two adanal shields in the male

**Type Species**—*Ceratixodes putus* Cambridge 1879 It lives on sea birds and is found on cliffs while *C. signatus* Banks 1908 is known in North America

me  
wl  
Capitulum long



FIGS 331 332—*Ixodes pilosus* KOCH 1844 VENTRAL ASPECT  
a Male (X 10) and mouth parts b mouth parts and female (X 10)

The female when young has a flat oval body. Replete female is like a castor-oil bean 10 to 12 by 6 to 7 millimetres of ashy colour. Tegument covered with fine short hairs. Dorsal surface with three well marked posterior grooves and two anterior.



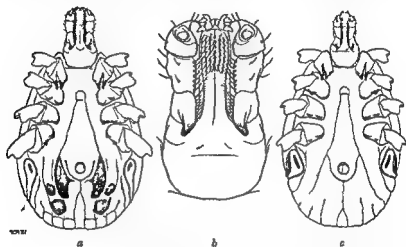
FIGS 333 335—*Ixodinus gervaisi* LUCAS 1847 VENTRAL ASPECT  
a Male (X 15) b mouth parts (more highly magnified) c female (X 15)

Genital pore at the level of the fourth coxae. Genital grooves unite in front of the vulva. Well marked, pre-anal crescentic groove open posteriorly. Stigmata whitish. Porose areas elongated.

***Hyalomma ægyptium* L. n.næus 1758**

**Synonyms**—*Acarus ægyptus* L. 1758 *Ixodes camelinus* Fischer 1823

This tick is found in Africa particularly in Egypt and South Africa in Asia particularly in Southern India in Europe especially in France and Italy. The adults attack cattle especially sheep and goats and also at times man. The larvæ and nymphæ are supposed to attack birds not cattle.



FIGS 339-341.—*Hyalomma ægyptium* LINNÆUS 1758 (×3) VENTRAL ASPECT  
 a Male (×8) b mouth parts (more highly magnified) c female (×8)

The male is almost black with a pale marginal stripe with a small triangular often white median festoon.

The female is brown with light blue stripes. Scutum which

**SUBORDER IV PROSTIGMATA**

The suborder Prostigmata contains two superfamilies which are

last  
high

alpi  
the



is commonly found in the South of England during August and September. It is also common in France during the hot and dry months. Bruyant raised a nymph in 1800 which was thought at first to be *M. pusillum* Hermann but this is now thought to be doubtful. It is also found in Germany. It generally attacks small mammals such as dogs and cats. *Acarus batatus* Linnaeus of Surinam perhaps belongs here. *M. wichmanni* Oudemans is found in New Guinea and Celebes.

**Microtrombidium akamushi** Brumpt 1910

**Synonyms** — *Ikamushi* (red mite) *Kedani* (hairy mite) *Slacht* (sand mite) *Shimamushi* (island mite) *Tsutsugamushi* (dangerous mite) *Yochubia*

**Definition** — *Microtrombidium* of various characters probably covering several distinct species with and without all dorsal hairs of the palp feathered. Hair on galea of maxilla always stout and feathered. Hairs on dorsum of palp not feathered except the tactile hair. Tarsal claw trifurcate. Last tarsus without long hairs.

**Remarks** — The form shown in Fig. 342 may be a distinct species from *M. akamushi* because all the dorsal hairs on the palps are feathered. This may be called *Microtrombidium brumpti*.

915

These are the mites which cause Japanese river fever (*Tsutsugamushi fever*).

**Morphology** — The larva is orange red in colour. 1002  
metre in length by 0.10024 millimetre in breadth. The legs are leg like and the body and legs are very hirsute.

The *scutum* is oblong not wide with straight posterior margin usually seven hairs. The pseudostigmata are nearer the anterior margin than the anterior margin.

Figs are well developed

D 1 2 8 ( has two  
1 1 1 1 1 1 1 1 1 1  
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♂ with a few posterior hairs. One hair on the galea of the maxilla which is feathered. The palps are stout rod like hair. Legs are 1002

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and fr  
lace  
larv

## SUPERFAMILY A TROMBIDOIDEA.

The Trombidoidea include the following families which are of importance in medicine—(1) Trombididae (2) Tetranychidae (3) Cheyletidae

## FAMILY TROMBIDIDÆ

Trombidoidea with soft skins and chelate mandibles adapted for biting

There are two important genera which may be differentiated as follows—

A Distal segment of palp with single claw—*Trombidium*

B Distal segment of palp with two claws—*Microtrombidium*

*Trombidium* Latreille 1795

The larva of this genus are the harvest mites and are widely distributed. *Leptus americanus* Riley and *L. irritans* Riley are American species being found in the United States and Mexico.

*Trombidium tlalsahuatl* Lemaire 1867 is the *Tlalsahuatl* of Mexico. The zoological names of the pou dogouti of Guiana the masbi of New Granada the colorado of Cuba the mouqui of Para the b te rouge of Martinique and Honduras are not known. It must be confessed that there is a great deal of uncertainty about the genus and species of these larvæ and the subject evidently requires revision.

**Morphology**—They are six legged larva with prominent claws on the tips of their legs provided with a powerful hypostome which they drive through the skin. Around this hypostome the tissues of the host are supposed to form a tube.

**Life-History**—Only the larvæ appear to be parasitic the adults apparently are not.

**Pathogenicity**—They cause itching, redness and swelling of the affected part which if scratched may become eczematous and even at times suppurate.

**Treatment**—Sulphur ointment kills them.

Genus *Microtrombidium* Haller 1882

**Definition**—Trombididae in which the distal segment of the palpus terminates with two stout claws.

**Remarks**—These mites are quite common. Thus *Microtrombidium ahamushi* Shaw 1799 is the harvest bug of England and



116 311—*Microtrombidium ahamushi*  
BRUNPT 1910

(After Tanaka from *Centralblatt für Bakteriologie I ser und Inf*)



**Acaropsis** Moquin-Tandon, 1863.

*Acaropsis mericouris* Laboulbène has been found in the human external auditory meatus.

## SUPERFAMILY B EUPOPOIDEA

## FAMILY BDELLIDÆ.

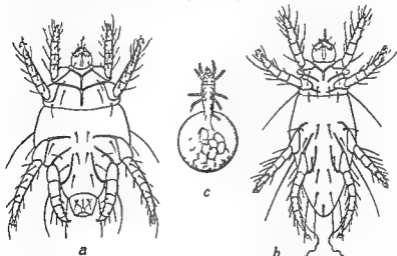
No specialized seta on cephalothorax, integument not chitinized or leathery; palpi composed of four or five segments, cephalothorax large and clearly separated from abdomen; palpi large, geniculate, and bearing distally long tactile bristles, mandibles chelate.

**Tydeus molestus** Moniez, 1889

This mite was imported into Belgium in Peruvian guano, and caused much trouble to man and beast.

## FAMILY TARSONEMIDÆ

With marked sexual dimorphism and tracheæ No ventral suckers  
*Tarsonemus hominis* Dahl found in cancerous tissues in man, is probably an accidental contamination of the preserving fluids



FIGS 343-3. —*Pediculoides ventricosus* NEWPORT.

a, Male ( $\times 350$ ), b, female ( $\times 220$ ), c distended female ( $\times 60$ ).

Genus *Pediculoides* Targioni-Tozzetti*Pediculoides ventricosus* Newport, 1850

**Synonyms.**—*Heteropus ventricosus* Newport, 1850, *Acarus tritici* Lagrèze Fossot and Montané, 1851; *Physogaster larvarum* Lichtenstein, 1868, *Pediculoides tritici* Targioni-Tozzetti 1878 *Spharogyna ventricosa* Laboulbène and Mégnin, 1885; *Tarsonemus monougniculosis*

This mite causes severe itching and urticarial eruptions on the breast

0.08 millimetre with six pairs of

and other vegetables After some growth the nymphs seek shelter

*Microtrombidium wichmanni* Oudemans 1905

Its larva attacks man and animals in Celebes

*Microtrombidium vandersandei* Oudemans 1905

Synonym — *Microtrombidium* Van der Sander

The larva of this Trombidium occurs in New Guinea and attacks man and animals Its local name is Gononc

*Metatrombidium* Oudemans 1909

Oudemans 1904 has been found on

TRANYCHIDÆ

Genus *Tetranychus* Dufour

Definition.—Tetranychidæ without cephalothoracic tubercles Few legs, legs longer than body which is not twice as long as broad Legs slender, legment not tessellated dorsally Palpi ending in a distinct thumb

*Tetranychus molestissimus* Weyenbergh 1896

FAMILY CHEYLETIDÆ

combs folded without spinous processes on the legs skin with few if any setae palpi much thickened at the base moving laterally last joint often with two pectinate bristles without eyes First leg ending in several long

*Cheyletes* Latreille 1796

These are very small mites distinguished by having enormous palpi with minute bristles *Cheyletes eruditus* has been described in the external nares of a man

sucking. The palpi are three jointed, and pressed to the under surface of the rostrum. The legs are eight in number short and consist of three segments with small terminal ungues. The abdomen is tapering striated dorsally and ventrally and rounded at the tip. The anus is situated at the anterior end of the abdomen.

**Demodex folliculorum** Simon, 1842

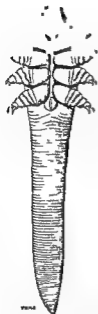


FIG. 35.—*Demodex folliculorum* SIMON 1842 (X 150)  
(Partly after Berlese.)

Male measures 300  $\mu$  by 40  $\mu$  and the female 360  $\mu$  by 45  $\mu$ .  
Life-History.—The eggs are 60 to 90  $\mu$  in length by 25  $\mu$ .

ARACHNIDÆ INCERTÆ SEDIS

LINGUATULIDA

*Parasitic Arachnoidea* with ringed elongated vermiform bodies possessing two pairs of hooks in the neighbourhood of the jawless mouth.

Remarks.—The *Linguatulida* have been found parasitic in man both in the adult and larval conditions but the adult is much rarer than the larva. They have been found in Europe and Africa and reported from the West Indies but this was in a negro from West Africa. It is not impossible that they will be found to be far from uncommon parasites when the medical history of the West Coast of Africa is better known. When dealing with cases showing obscure abdominal or

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ids

**Nephrophages Miyako and Scriba 1893**

*Nephrophages sanguinaris* Miyako and Scriba 1893 It is a very doubtful parasite of man it was found in bloody urine passed by a man in Japan

**SUBORDER V. ASTIGMATA,**

This suborder includes the superfamily Sarcoptoidea

**SUPERFAMILY SARCOPTOIDEA**

Astigmata with small three pointed palps adhering for some distance to the hypostome with usually ventral suckers Two families concern us— (1) Tyroglyphidae (2) Sarcoptidae

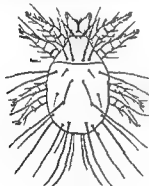


FIG 346 — *Tyroglyphus longior*  
VAR *castellanii* HIRST 1912  
DORSAL ASPECT

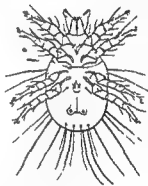


FIG 347 — *Tyroglyphus longior*  
VAR *castellanii* HIRST 1912  
VENTRAL ASPECT

**FAMILY 1 TYROGLYPHIDÆ**

Very small mites without eyes only accidental parasites being found in flour sugar cheese etc

*Tyroglyphus* sp

*T. longior* var *castellanii* Hirst 1912 was found by Castellani in copra and on people affected by copra itch in Ceylon In this variety in contrast to *T. longior* there is no pair of short hairs on the ventral surface behind the anterior suckers

*Glyciphagus* Hering 1835 — With dorsum covered with hairs

*G. prurorum* Hermann (synonym *G. domesticus* de Geer 1803) is the cause of grocer's itch

*Rhizoglyphus* Claparède 1860 — *I. parasiticus* Dalgetty 1911 — With short legs armed with spines Tarsi end in a claw Live on plants

A succession of ecdyses results in the formation of the nymph which resembles the adult except that it possesses numerous chitinous spicules on its skin and by the sixth to the seventh month is about 6 to 8 millimetres in length

These nymphæ now wander about the host and are supposed to reach the bronchi and thus to leave the herbivorous host and reach the dog either by the nose or mouth in the nasal cavities of which they moult and become sexually mature copulating about the sixth to seventh week after infection

**Pathogenicity**—Both the larva and the adult may be found in man the former in the lung rarely in the liver spleen or intestinal

America

**Porocephalus Humboldt 1811**

1811 1811 1811 1811 1811

work in parasitology extends

**Pathogenicity**—They cause porocephalosis in man and animals

**Porocephalus armillatus Wyman 1847**

stc Wyman 1847 *Penta*  
 m *moniformis* Neu  
 18 *diesingeri* Beneden  
 str ddell 1863 *P con*  
 1883 *Inguatulida*

*constricta* Kuchenmeister 1855

*Porocephalus* with cylindrical body slightly flattened on its anterior face and surrounded by about sixteen to twenty two distinct rings separated from one another by a wide interval The

*P. reclus* *Bitis nasicornis* and *B. arietans*) in the individual in marmoset and other hedgehog and in

The sexes are distinct. The mouth leads into a simple straight alimentary canal which ends in the anus. There are no circulatory or respiratory organs. The nervous system consists of a ventral mass and a circumoesophageal commissure.

**Life-History**—The female produces eggs which, escaping from the definitive host again.

**Genera**—*Linguatula* Frolich 1789 *Porocephalus* Humboldt 1811, *Reighardia* Ward 1899 and *Rasilictisella* Sambon 1909 but only the first two contain species parasitic in man.

#### *Linguatula* Frolich 1789

*Linguatulida* with depressed body, rounded dorsum and crenate margins. Body cavity forming diverticula into the lateral parts of the rings.

**Species**—*Linguatula serrata* Frolich 1879

#### *Linguatula serrata* Frölich 1789

**Synonyms**.—*Tania rhinaria* Pilger 1802 *Polystoma lanioides* Rudolphi 1810 *Linguatula lanioides* Lambinet 1816 *Pentastoma lanioides* Rudolphi 1819 **Nymph**—*Pentastoma denticulatum*

The adult lives in the nasal cavity and frontal sinus of the dog, wolf, fox and rarely in the horse, mule, sheep, goat and man in Europe, while the larva exists in sheep, oxen, horses, rarely in cats and dogs. Its real host appears to be the dog, especially sheep dogs.

**Habitat**.—Europe, especially Central France.

**Morphology**—The male is white in colour, 18 to 20 millimetres in length and 3 millimetres broad. The female is greyish white or yellowish.

90 by 70  $\mu$  are laid in

perforating apparatus composed of a stylet and two hooks.

It now bores its way into the liver, lungs or some other organ and in about eight weeks becomes encysted, losing all its appendages and measuring 275 by 180  $\mu$ .

merely encysted in the lungs and liver but moving freely through the peritoneal cavity and in the small intestine Sambon considers that the eggs pass from the snake into water and thence into animals



FIG 357 — POSTERIOR END OF *Porocephalus armillatus* WYMAN (X 5)  
(After Sambon)



FIG 358 — NYMPH OF *Porocephalus armillatus* WYMAN ENCYSTED IN THE LIVER.  
(After Sambon from our West African case)

and man while drinking and become larvæ and nymphæ which later gain access to the snake when the host is killed and eaten

Pathogenicity — This will be described later (Chapter LXXXIII)

#### *Porocephalus moniliformis* Diesing 1836

Synonyms — Adult — *Pentastoma moniliforme* Diesing 1835 <sup>P</sup>  
*moniliforme* Leuckart  
*Porocephalus monilifor*  
*tornatus* Creplin 1849

*Porocephalus armillatus* Stiles *pro parte*

*Porocephalus* with twenty six to thirty one rings

Remarks — This parasite which was discovered by Czermak in 1872 and was first described by Sambon who *atus* in general ap-  
be easily mistaken

for it

Morphology — It is more slender tapers more caudad with twenty six rings in the male and twenty-eight to thirty one rings in the female In fresh specimens it is bright lemon yellow in colour with genital opening on the mid ventral surface of the first body ring in the male and on the mid ventral surface of the terminal body cone 1 millimetre in front of the anus in the female The anus

Linnaeus (the  
lated python)  
*nymph* are man  
(?)  
the Philippines

**Morphology**—Female ♀ to 12 centimetres in length and 5 to 9 millimetres in breadth with eighteen to twenty two rings each 1 to 2 millimetres in width. The cephalothorax extends from the anterior end of the body to the first body ring which is often very indistinct. Dorsally this region is convex while ventrally it is concave and carries the mouth in front of which there are two



FIG. 353—*Porocephalus armillatus* WYMAN MALE NATURAL SIZE (After Sambon)



FIG. 354—*Porocephalus armillatus* WYMAN FEMALE NATURAL SIZE (After Sambon)

papillæ and on either side of which there are two hooks. Genital opening about 1 millimetre in front of the anus which is terminal. Male 3 to 4.5 centimetres in length and 3 to 4 millimetres in breadth with sixteen to seventeen rings. Genital opening in the middle



FIG. 355—LATERAL ASPECT OF THE CEPHALOTHORAX OF *Porocephalus armillatus* WYMAN (X5) (After Sambon)



FIG. 356—VENTRAL ASPECT OF THE CEPHALOTHORAX OF *Porocephalus armillatus* WYMAN (X5) (After Sambon)

of the ventral surface at the anterior end of the abdomen.

**Life-History**—Probably this resembles that of *Lyneborgia ulva serrata* with the difference of habits. In man the nymphs are found not





**Pathogenicity**—So far only two cases have been recorded in man—one at Djambi in Sumatra in 1906 when a nymph was found encysted beneath the serous coat of the small intestine of a Djambi native who died of dysentery and the other was found in the liver of a native Filipino who died of tuberculosis. The liver in this latter case showed signs of atrophic cirrhosis.

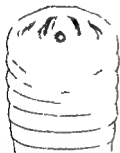


FIG. 359.—VENTRAL ASPECT OF THE CEPHALOTHORAX OF *Porocephalus moniliformis* DIEBING 1836 (X 5)



FIG. 360.—*Porocephalus moniliformis* DIEBING 1836 FEMALE NATURAL SIZE

(After Sambon)

### Species imperfectly described in Man

In addition to the well known cases of porocephalosis due to *Porocephalus armillatus* and *P. moniliformis* there are the following cases to be discussed—viz Welch's parasite Oeler's parasite and Flint's parasite. Oeler's parasite is considered to be doubtful as it was passed *per urethram* and might according to Sambon have been a sparganum.

#### Welch's Parasite

In the *Lancet* of November 16 1872 F. H. Welch had an article on "The Presence of an Encysted Lechnorhynchus in Man." This parasite was damaged in extraction and his drawing was not very instructive but was sufficient to convince Cobbold R. Blanchard and Sambon that it represented a Linguatulid and it shows two sets of hooks. The last named observer comes to the conclusion that it may be either a very early nymph of *Porocephalus moniliformis* (provided with caducous accessory hooks) or it may be *P. rufus* Leuckart 1860 (which is found in the abdominal muscles and peritoneum of the cobra) or *P. erectum* Parona 1869 (found in *Crocidura fuliginosa* a musk shrew) or it may be a new species.

walking legs one pair to each somite Body anomomeric showing from 17 to 175 somites behind that which carries the poison gland They breathe by tracheæ and the genital ducts open on the penultimate somites

Family 1 Scolopendridæ

Family 2 Lithobiidæ

Family 3 Scutigeraidæ

#### FAMILY 1 SCOLOPENDRIDÆ Leach 1812

*Chilopoda* with antennæ possessing few joints and with few ocelli

*Geophilus carphophagus* Leach *G electricus* Linnæus *G cephalicus* Wood *G similis* Leach have been found as accidental parasites about nineteen times in the nasal cavities and their neighbouring sinuses in man in Europe *G electricus* Linnæus has been found in the alimentary canal about four times

#### FAMILY 2 LITHOBIIDÆ Newport 1844

*Chilopoda* with many jointed antennæ numerous ocelli

*Lithobius fortificatus* L and *L melanops* have been found in the nasal cavities in three cases in man

#### FAMILY 3 SCUTIGERIDÆ Guvial 1837

*Chilopoda* with antennæ at least as long as the body and faceted eyes instead of ocelli

*Scutigera coleoptrata* has been found in the alimentary canal

Other species found in the alimentary canal are *Chatechelys vesuviana* Newport (found also in the nasal cavities) *Humanitaris in geraisi* *Stigmatogaster subterraneus*

**Pathogenicity**—In the nose these parasites cause inflammation with at times no flow of mucus and at others a large discharge of it associated with headache which is generally more or less continuous but may show remissions In addition to these local symptoms general symptoms such as convulsions anguiform attacks dyspnoea etc may be induced through irritation of the fifth nerve There is no evidence that these parasites cause any of the

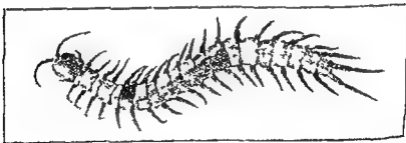


FIG 362—SCOLOPENDRA SPECIES (?)

(This is a very common species in Ceylon)

generally expelled in attacks of sneezing making them leave the nostrils are turpentine but in some instances be frontal sinus—by surgical means

will give rise to the suspicion of abdomen cramp nausea vomiting

and reflex nervous symptoms

Treatment does not appear very satisfactory

## CLASS IV CRUSTACEA LAMARCK 1815

*Aquatic Arthropods* which breathe by means of gills

Crustacea is can hardly be considered as human parasites for they have very rarely occurred as such—e.g. *Calig* is a *ectoparasite* in the cornea and *Gammarus pulex* in the stomach.

## ORDER COPEPODA Latreille 1831

It must however be remembered that the Copepoda are of importance



FIG 361 -CYCLOPS SP (?)

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hours to raise 87 gallons of water as steam is c 12 square feet of grate & ve  
90 gallons of water as steam in half an hour

## CLASS V CHILOPODA LATREILLE 1831

*Arthropods* with three pairs of legs. The first post-oral somite is the mandibular, the second and third post-oral somites carry the maxillae, while the fourth has its appendages converted into very large powerful jaws which are provided with poison glands. The remaining somites carry 7 pairs of legs.

**Sarcoptidæ.**

- CASTELLANI (1906) *Centralblatt für Bakteriologie*  
 HIRST (1917) *Arachnida and Myriopoda Injurious to Man* British Museum  
 (Natural History)

**Demodidæ.**

- BENTARELLI AND PARAMIOS (1911) *Centralblatt für Bakt* Jena Abt 1

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- CHALMERS (1899) *Lancet* 1 January 1  
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 Leipzig  
 SAMRON (1910 1912) *Journal of Tropical Medicine and Hygiene* London  
 (A very excellent account of the parasites found in man and animals)  
 SHIPLEY (1898) *Archives de Parasitologie* 1 52

**Chilopoda**

- BLANCHARD (1898) *Archives de Parasitologie* (1910) *Archives de Parasitologie*  
 LAVERAN AND ROUBAUD (1916) *Bull Pathol Exot* vol 11 p 64  
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## Acarina

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English edition

## Ixodoides

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DUTTON AND LODD Liverpool School of Tropical Medicine Memoir XVII  
(*O. mombata*)

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Memoirs Soc Zool France IX 189 (Argasidae) X 1899 (Rhipicephalidae)

XII 1901 (Ixodinae) XIV (Summary Classification Tables)

II 451 1902 Archives de Parasitologie VI 194 VIII 1905 IX

1906, X 1907 Annals of Tropical Medicine and Hygiene  
Berlins

Journal 11 1909 1147 (Ticks and  
Hospital Reports VIII

Cambridge (A most useful monograph) also many papers in the  
Journal of the

3 152

17 of April

(Genus

## Akamushi.

MIYAJIMA AND OKUMURA (1917) *Kansu* & Archives of Experimental  
Medicine 1 1 April

## Tyroglyphidae

HIRST (1915) Journal of Economic Biology x 4 (The Harvest Bug and  
the Akamushi)

OLDMANS (1911) Oudemans Gravenhage Ber Med Ent Ver Juss

PEPPER SCHMIDT AND SMITH (1908) University of Pennsylvania Medical  
Bulletin

- The exoskeleton of the head is composed of sclerites—that is to say more densely chitinized regions of the integument—which are (1) The clypeus (2) the epicranium (3) the gula

The clypeus is the sclerite situated on the anterior portion of the dorsal surface and carrying the labrum in flies it is often called the face

The epicranium is the larger part of the head and may be subdivided

styliform organs



FIG. 364.—WING OF A MOSQUITO TO ILLUSTRATE THE VENATION  
(From a photograph by J. J. Bell)

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## CHAPTER XXIX

### THE HEXAPODA

Synonym—Remarks—Morphology—Internal anatomy—Life-history—Habitats  
 —Enemies—Pathogenicity—Collection—Classification—References

**Synonym—***Insecta*—Arthropods breathing by means of tracheæ with antennæ on the head three pairs of legs and usually two pairs of wings on the thorax, which is composed of three segments. Abdomen with generally nine apparent segments.

**Remarks—**The Hexapoda or insects are known to be of the utmost importance in the spread of disease for the researches of Manson, Ross, Grassi and others have shown that they are agents in the propagation of the parasites of filarial malaria and other diseases.

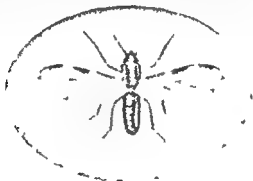


FIG. 363.—*Culicoides trucei*. A TYPICAL DIPTEROUS INSECT  
 (From a photograph by I. J. ReB.)

<p><b>Morphology</b>—The head is small and rounded, with two large compound eyes and a pair of antennae. The thorax is broad and bears two pairs of wings. The abdomen is segmented and tapers to a point.</p>	<p>of abdomen          the hind          see next          last but          doubtful          one of          premaxillary          maxillary          pair of</p>
<p>maxillæ usually fused to form the upper lip or labrum with a pair of labial palps.</p>	<p>generally carries</p>



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styluom the first pair of maxillæ, similarly may be broad, strong organs or styliform organs



FIG 364 —WING OF A MOSQUITO TO ILLUSTRATE THE VENATION.  
(From a photograph by J J Bell)

while each pleuron is divided into an anterior episternum and a posterior epimeron. According to Audouin a typical thoracic segment should have a notum composed of præscutum, scutum, scutellum, and post-scutellum.

13 Costal Cell between the Coxa and the Subcoxa

- 14 First Anal between Cubitus 2 and Anal 1  
 15 Second Anal between Anal 1 and Anal 2  
 16 Axillary between Anal 2 and Anal 3  
 17 Spurious Cell behind Anal 1

But all these cells are not present in any one given type of wing owing to coalescence of the same.

larynx, and so opens near the tip of the proboscis.

From the mouth a pharynx leads through an oesophagus with a dilatation called the crop into a proventriculus or rasticatory stomach, which latter communicates with the mesenteron or chylific ventricle whose junction with

- the intestine is defined by the openings of the caecal Malpighian tubules. This is an important landmark defining where the stomach ends and the intestine begins. The intestine is subdivided into small intestine, colon or

6.113



FIG 365—*Phthirus pubis* LINNÆUS 1758 TO ILLUSTRATE AN INSECT WITHOUT WINGS  
(From a photograph by J J Bell)

The stigmata or openings of the respiratory system are situated on the

lateral vessels and one dorsal and one ventral vessel. The blood is colourless and contains amoeboid cells

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1 Costal Cell between the Costa and the Subcosta

- 14 First Anal between Cubitus 2 and Anal 1
- 15 Second Anal between Anal 1 and Anal 2
- 16 Axillary between Anal 2 and Anal 3
- 17 Spurious Cell behind Anal 1

H + all these

on the hypo  
 in a dilatation  
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 juncture with

- species. This condition of life which is exactly similar for many different

the genera and families are but few in number. Thus for example in the Mallophaga there are about 1500 known species grouped into 27 genera, 4 families and 2 suborders.

The important difference between the Mallophaga and the Anoplura is that the former have a masticatory mouth while the latter have a sucking mouth.

#### ORDER MALLOPHAGA Nitzsch 1818

Synonyms — *Mandibulata* De Geer 1763 *Ricinidae*

Nomenclature — The name Mallophaga is derived from *μαλλος* wool and *φαγε* to eat.

Definition — Hexapod, usually of small size, wingless, provided with biting mouth parts and with simple incomplete metamorphosis. Habitat

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 a Plate 2 called Pulino  
*ilus columbae* or pigeon  
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The prothorax is distinct but the meso- and meta-thoraces are sometimes united and may also be with difficulty differentiated from the abdominal segments. The legs are flattened, long and strong and end in one or two claws.

The body varies in colour, being whitish, pale brownish or dark brown in colour and marked by darker spots and bands which are caused by chitination.

The pharyngeal sclerite may be present or absent, as may be the accompanying glands; the crop may be simple or have a sac-like diverticulum. Inguinal

which is a vagina. This receives the cement glands and the sebaceous glands which provide the secretion for gluing the eggs together and the receptaculum seminis beneath which is the bursa copulatrix which is sometimes separated from the vagina.

**Life-History**—This is very varied. In general terms the spermatozoa bound into bundles of spermatophores are introduced by the external copulatory organ which when protruded is surrounded by the spermatheca.

Common forms

Usually the egg develops into a larva which becomes a nymph or pupa and this into an adult often called the imago. This series of changes is called a metamorphosis. The pupa may present three forms:

1. *The free pupa* with appendages free
2. *The oclept pupa* with appendages and body bound together
3. *The coarctate pupa* in which the pupa is contained in a puparium formed from the larval skin.

The larva is a most vigorous feeder but the pupa does not take

sucking insects. As a rule only the female sucks blood which apparently it does with a view of obtaining rich nourishment for its eggs. It is this habit which makes these insects of importance in tropical medicine because not merely do they cause irritation but

may cause disease.

**Enemies**—Insects and their larvae have many enemies in birds, reptiles, fish and other insects. Cannibalism is also met with especially among mosquito larvae.

**Pathogenicity**—The principal disease spreading insects are the Diptera—for example the *Anophelinae* disseminate malaria, the genus *Stegomyia* yellow fever and the genus *Culex* filariasis and are capable of leading the

in the dissemination of typhoid and perhaps dysentery in tropical countries. Fleas are now known to be the spreaders of plague.

## ORDER ANOPLURA Leach, 1815

**Synonyms.**—*Pediculina* Burmeister, 1835, *Siphunculata* Meiner 1891; *Pseudorhynchota* Cholodkowsky, 1903, *Lipognatha* Börner 1904; *Ellipoptera* Shupley, 1904

**Definition.**—Hexapoda with labrum and labium joined together to form a rostrum or proboscis, which is armed with recurved hooklets, and contains a hollow extensile sucker formed by the mandibles and maxillæ. Eyes without facets. Antennæ five jointed. Thorax with little traces of segmentation. Wings absent. Legs with hook-like terminal joints, suitable for clinging. Last abdominal segment rounded in male, notched in female. Metamorphosis incomplete. Habitat, epizotic on mammals.

With reference to the classification of the lice, see the notes for Aristotle.

**Morphology.**—The anatomy will be dealt with under the heading *Pediculidæ*, and need not detain us here, except to invite attention to the characters of the mouth parts, which are so modified as to form a sucking mouth, while the pharynx performs the rôle

Classification. The lice are divided into three families, some of which are all. The number of segments of the antennæ, which are all five, is the character to be differentiated —

A Head not prolonged into a nozzle-like projection. Antennæ three to five segments. Tibia with a thumb-like process. Tibia and tarsus very short and thick. Legs clinging in character.

I Body flattened. Mesothorax and three to eight abdominal segments, with stigmata. Antenna three to five segments. Tibia with thumb like process.

have been found to be parasitic on about 100 species of mammals representing 46 genera 24 families and 5 orders and 1100 bird species or 33 orders of

has influenced the evolution each host being according to is made up of many dislocated as to be given a very flexible

and family few

Hence some 1500 species are known which can be divided into two sub

and 10 genera

The following table gives the differentiation of the suborders families —

#### SUBORDER 1 ISCHNOCERA KELLOGG 1896

Antennæ exposed filiform three or five segmented no maxillary palpi mandibles vertical crop with sac like diverticula ingluvial glands present testes four egg tubes five

A Antennæ three jointed tarsi one claw Habitat mammals—*Trichodectidæ*

B Antennæ five jointed tarsi two claws Habitat birds—*Phlopteriidæ*

#### SUBORDER 2 AMBYCERA KELLOGG 1896

Antennæ concealed clavate or capitate four segmented maxillary palpi present mandibles horizontal crop single ingluvial glands absent testes six egg tubes three to five

A Tarsi with one claw Habitat mammals—*Gyropidæ*

B Tarsi with two claws Habitat birds—*Liotheridæ*

The genera parasitic on mammals may be recognized as follows —

#### SUBORDER ISCHNOCERA KELLOGG 1896

##### FAMILY TRICHODECTIDÆ Burmeister 1845

This family contains only one genus *Trichodectes* Nitzsch 1818 which is parasitic on mammals and has tarsi with only one claw

#### SUBORDER AMBYCERA KELLOGG 1896

##### FAMILY GYROPIDÆ Burmeister 1835

This family contains only one genus *Gyropus* Nitzsch 1818 parasitic on mammals and with tarsi armed with only one claw



The alimentary canal consists of a mouth followed by a chitinous pharyngeal pump into which the ducts of the salivary glands open and an oesophagus.

ovary consists of five to six follicular tubes. The two oviducts unite into a common duct and there is a spermatheca. The male organs are testes, vasa deferentia, vesiculae seminales, and penis.

**Life-History.**—The ova which are attached to the hairs of the host, give rise to larvae which closely resemble the adults.

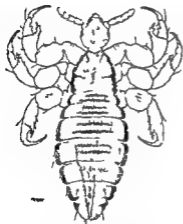


FIG 366 — *Pediculus humanus*  
L. MALE (X 25)

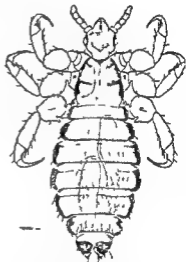


FIG 367 — *Pediculus humanus*  
L. FEMALE (X 25)

**Pathogenicity.**—Lice are important carriers of disease, being vectors in certain forms of relapsing fever in typhus, and in trench fever, etc.

**Classification.**—The Pediculidæ are subdivided into two subfamilies, which may be recognized as follows—

A *Pediculidæ* with five-jointed antennæ—*Pediculinæ*

B *Pediculidæ* with three-jointed antennæ—*Phthirinæ*

#### SUBFAMILY A. PEDICULINÆ Enderlein, 1904

D. St. (H. n.)

Cl. (St. (H. n.))

(a) All legs strong, distinct neck, thorax narrower than abdomen—*Pediculus*

(b) Forelegs long, slender, with three claws, no neck, thorax broader than abdomen—*Phthirus*

(a) Head broader than thorax Eyes large pigmented Pharynx short and broad Proboscis short and pressed against thorax—*Pediculidæ* Leach 1815

(b) Eyes very small or absent Pharynx long and narrow Proboscis very long—*Hæmatopinidæ* Enderlein 1904

II Body thick and heavy Mesothorax metathorax and two to eight abdominal segments with stigmata Eyes absent Back part of the head widened backwards Antennæ four to five segments Tibia with short strong thumb like process Thick short spines on the body Female gonopodia elongated and narrow—*Echinophthiridæ* Enderlein 1904

B Head prolonged into a nozzle like projection at the anterior end of which lies the mouth opening Antennæ five segments Tibia without thumb like process Tibia and tarsus very long and thin Legs not clinging in character—*Hæmatomyzidæ* Enderlein 1904

#### FAMILY I PEDICULIDÆ Leach 1815

**Definition**—Anoplura with flattened body and head not pro

**Remarks**—The Pediculidæ are found all over the world on man as well as on animals They cause much irritation by their bites which may become

† *unus pediculi*  
Morphology Th. 2

the legs have to  
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of se  
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It

marked by lateral festoons. Thumb like projection on the tibia armed with a spine. Habitat homo.

These pediculi are extremely common in the tropics and it is an every day heads  
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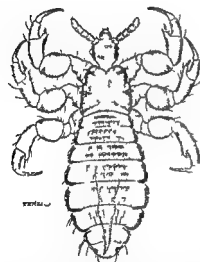


FIG 369—*Pediculus corporis* DE GEER MALE (X25)

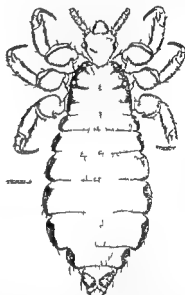


FIG 370—*Pediculus corporis* DE GEER FEMALE (X25)

**Male**—The posterior somite is rounded off and prominent with a circular opening dorsally which is the common aperture of the genital and alimentary canal. The penis is simple wedge shaped and is usually seen protruding dorsally.

days

**Pathogenicity**—It can carry typhus and produce a form of impetigo.

**Treatment**—White precipitate ointment or common paraffin oil may be used.

Genus *Pediculus* Linnæus 1758

**Definition.**—Pediculæ with distinct neck and thorax narrower than abdomen. Legs all strong with strong claws. Thumb like.

Female gonopodia clamp like and bent inwards.

**Classification.**—Only four species are at present assigned to the genus *Pediculus*—

There is a doubt as to whether *P. punctatus* is really a pediculus and *P. consobrinus* is said by Neumann to be indistinguishable from *P. humanus* therefore the species are reduced to two—

*P. humanus* Linnæus 1758

*P. corporis* de Geer 1778

These are very alike and have been thought to be only varieties of one another. Interbreeding seems to be possible. The following points may help to differentiate them—

- 1 Found on the human head with well defined abdominal segments marked by a festooned lateral border—*P. humanus*
- 2 Found in the clothing larger than *P. humanus* with broader thorax and lateral borders of abdomen less festooned and segments not quite so distinctly indicated—*P. corporis*



FIG 368.—EGG OF *Pediculus humanus* LINNÆUS 1758 ATTACHED TO A HAIR (X35 DIAMETERS)

(From a photograph by J. J. Bell)

*Pediculus humanus* Linnæus 1758

**Synonyms.**—*Pediculus humanus* var. 1 Linnæus 1766 *P. humanus* var. *capitis* de Geer 1778 *P. cervicalis* Leach 1817 *P. capitis* Nitzsch 1818 *P. humanus* Csiki 1904 *P. nigritarum* Latreille

**Definition.**—*Pediculus* often varying in colour somewhat according to the human race on which it is parasitic with thorax often narrowing distinctly anteriorly well defined abdominal segments



**Pediculus corporis de Geer 1778**

Synonyms.—*P. humanus* Linnæus 1758 *P. humanus* var. ■  
Linnæus 1766 *P. humanus* var. *corporis* de Geer 1778, *P. testis*  
*menti* Nitzsch 1818 *P. corporis* Csiki 1904

Definition.—Pediculus usually dirty white in colour thorax  
only slightly narrowed anteriorly abdominal segments not very  
distinctly defined and not well festooned laterally Thumb like

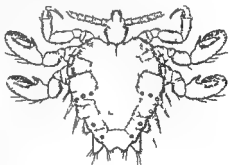
Life-History—It lays seventy to eighty eggs measuring 0.7 to  
in three to

I  
and certain forms of relapsing fever

Treatment—Boil or steam the clothes in a sterilizer at 212° F

**Genus Phthirus Leach 1815**

Pediculidæ with the anterior legs weak armed with large short  
claws abdomen broad and short with first to fifth segment so  
strongly compressed that the stigmata appear to lie  
in one segment Fifth  
to eighth segments with  
lateral tooth like pro-  
cess of which the two last  
are long Habitat homo



*Phthirus pubis* Lin-  
næus 1758

Synonyms.—*Pediculus*  
*pubis* Linnæus 1758  
*P. inguinalis* Reichard

also be found on the eyelashes and on the eyebrows

Morphology—The body in general is flat and broad with a



1817 is found on *Sus scrofa* Linnæus, other species are found on equines, bovines, camels antelopes deer, coluges, and monkeys

SUBFAMILY LINOGNATHINÆ Enderlein, 1904

The subfamily consists of five genera with the following characters—

1817

II Abdomen more or less elongated Pleura two to six provided with a dorsal and ventral long keefe like pointed process Four to seven tergites and sternites composed of three secondary segments each with a transverse row of long broad scale like hairs third tergite and sternite composed of two rings each Anterior ring is broader than posterior—*Hoplopleura* Enderlein 1904

B Abdomen without sclerites with smooth border Hind legs same size as middle leg —

I Each sternite and tergite of the abdomen with two or three transverse rows of very long closely set hairs—*Linoognathus* Enderlein 1904

II Each sternite and tergite of the abdomen with one transverse row of hairs —

(a) Stigmata large and those on the abdomen raised like

(b)

The genus *Polyplax* contains a rapidly increasing number of species The one illustrated in Fig 172 is *P. spinulosa* (Burmeister 1819) which is found on *Epimys noronensis* Erxleben 1777 in Europe

SUBFAMILY C EUHÆMATORININÆ Enderlein 1904

This subfamily has two genera with one species and this can be recognized as follows —

A Hind legs with femur and tibia armed with projecting rectangular, stalked sheath like appendages—*Euhæmatorinus* Osborn 1896

Species *E. abnormis* Osborn 1896 on *Scalops aratus* in North America

B Hind legs normal—*Hæmatopisoides* Osborn 1891

Species *H. squamosus* Osborn 1891 on *Geomys burmanni* in North America

REMAINING FAMILIES

The family *F. l.* contains three genera—  
 species *Tachinophthirus* Enderlein 1904 with only  
 attains one genus *Hæma* on the Ceylon elephant



## GYMNOCERATA FIEBER.

The families of importance to us are:—

- Clinocoridae  
 Reduviidae Stephens  
 Aradidae  
 Hydrometridae

These families can be diagnosed, according to Distant, in the following manner —

- A Species not aquatic, abdomen not clothed beneath with a silvery, velvety pubescence, scutellum not reaching to the base of the membrane nor to the middle of the abdomen
- (1) Mesopleuræ and metapleuræ composed of one piece, hemelytra without cuneus
- (a) Tarsi three-jointed. Rostrum short, stout, bent at the base, so that in repose it does not lie against under-surface of the head, ocelli behind eyes, hemelytra complete with distinct membrane—*Reduviidae*
- (b) Tarsi two-jointed, anterior legs normal and inserted on the disc of the prosternum Hemelytra neither reticulate nor cellular—*Aradidae*
- (2) Mesopleuræ and metapleuræ composed of seven pieces, hemelytra with a cuneus and an embolium, ocelli absent—*Clinocoridae*
- B Species aquatic or semi-aquatic, abdomen clothed beneath with a silvery velvety pubescence, antennæ four-jointed—*Hydrometridae*.

## FAMILY CLINOCORIDÆ

Synonym.—*Acanthiadae*

Definition.—Gymnocerata without ocelli with elytra so short that the abdomen is not covered. Tarsi three-jointed

Remarks.—*Cecius* Stal, 1873. *Loxaspis* Rothschild, 1912, *Calocanus* Stal, 1873. *Bertilia* these are parasitic on bats

## CLINOCORINÆ

*Clinocoris* Petersonn, 1829

*Cecius* Linnaeus 1758, *Acanthias* Fabricius, 1803.

*Cismex* Phny.

CHAPTER XXXI  
HEMIPTERA

Hemiptera — Gymnocerata — Clinocoridae — Reduviidae — Hydrometridae —  
Aradidae — Reference

ORDER HEMIPTERA Linnæus 1742

Synonyms — *Rhyngota* Fabricius *Rhynchota* Burmeister

Definition — Hexapoda with four wings the front pair being either membranous or half horny and half membranous but both pairs may be wanting in the parasitic species Mouth suctorial Metamorphosis complete

Remarks. — The Hemiptera include the cochineal insect *Coccus cacti*, the Aphidæ plant lice and in particular the cicadas whose shrill notes wake the quiet of an African forest The anterior wings are called hemelytra and usually consist of three portions —

1 The clavus—the hard coriaceous portion next to the scutellum

2 The corium—hard coriaceous portion occupying the whole of the area between the clavus and the membrane

3 The membrane—apical portion

The posterior pair are the true wings

In the Clinocoridae the corium is divided into three portions —

1 Internal—corium proper

2 External and basal—cubulum

3 External and apical—cuneus

Classification. — The order is divided into two suborders —

SUBORDER I. HOMOPTERA — With both pairs of wings membranaceous

SUBORDER II. HETEROPTERA — With the front pair of wings half horny

The first will not be considered here

HETEROPTERA.

lunar, with two rounded horns. The dorsum is raised in the median line. The mesothorax is triangular, with the apex posteriorly. The metathorax is covered dorsally by the elytra, which are two small chitinous plates belonging to the mesothorax, but consist of clavus, corium, empodium, cuneus, and membrane. The abdomen, which is rounded in shape, is broadest at the posterior end. The male is smaller than female, with penis flexed into a notch between seventh and eighth segments.

**Internal Anatomy.**—The mouth leads into the pharynx, beneath which is a syringe organ or salivary pump, into which the salivary glands open. The pharynx leads via the oesophagus into a large crop. There is the usual mid-gut, intestine and rectum.

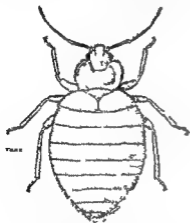


FIG 373—*Clinocoris lectularius*  
L. MALE (X10)

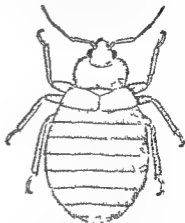


FIG 374—*Clinocoris lectularius*  
L. FEMALE (X10)

**Life-History**—They live in cracks in floors, walls, and furniture during the winter. They suck blood from human beings from house to house about four or five times a year. Complete development is said to require eleven weeks, but may be completed in as short a time as seven weeks.

The eggs, which are oval, 1-12 millimetres in length, and white in colour, take about seven to ten days to hatch. The larva grows slowly, moulting about five times at intervals of about eight days, after which the wing pad appears, showing that the adult stage is reached. They feed only upon blood.

**Bionomics**—*Bite*—In biting, they extend the proboscis and feel about, testing the skin with the delicate hairs, then pierce it by the stylets, and inject saliva by the syringe, causing congestion of the area when the blood runs up the grooves in the stylets by capillary attraction into the pharynx.

*campestris* which is found under stacks of corn in Europe may find its way into barns and stables and will then attack horses and cattle though under normal circumstances it would merely suck

*C. persleræ*

*C. boneti* Brumpt 1910 of our previous editions becomes *Leptocimex boneti* (Brumpt 1910) and *C. inodorus* of previous editions becomes *Hæmatosiphon inodorum* which is found on fowls and may enter dwellings

*Clinocoris lectularius* Linnaeus 1758

**Synonyms**—*C. lectularius* Merrett 1667 *Cimex lectularius* Linnaeus 1758 *Acanthisa lectularius* Fabricius 1794

**Definition**—*Clinocoris* with short broad head with two prominent eyes but no ocelli antennæ four jointed apical joints slender

have an ... ..

colour with short broad head carrying two eyes two antennæ composed of four segments of which the first and second are stout and the third and fourth slender The mouth consists of a proboscis which is composed of an upper part—the labrum—which is small and a lower curved portion This is large and jointed—the labium—inside which are four stylets the two outer being the mandibles and the two inner the maxillæ There are no palpi The prothorax is semi

**Climocoris rotundatus** Signoret, 1852

**Synonyms**—*Cimex rotundatus* Signoret 1852 *Cimex macrocephalus* Fieber 1861 This is the Indian bed bug which is found in India Ceylon Burma Assam and Malaya in Asia and also in Sierra Leone Mauritius Reunion St Vincent and Porto Rico

**Morphology**—*C. rotundatus* differs from *C. lectularius* by being darker in colour with a shorter narrower head with a prothorax

**Climocoris ciliatus** Eversmann 1841

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**Pathogenicity**—Not known

**Genus Cæcius** Stal 1873

e thorax slightly excavated and

1839 also *Cæcius vicarius* in

**Loxaspis** Rothschild 1912

*Climocorida* with anterior margin of thorax very narrow scutellum trans

( ) ( ) " " " "

**Genus Cæcodinus** Stal 1873

*C. villosus* on Natal Transvaal and Nyassaland bats *C. senotus* on African bats *C. indicus* on Indian bats

**Genus Aphramia** Champion 1900

*Aphramia barys*

**Genus Hæmatesiphon** Champion 1900

*H. inodorum* usually found on fowls but may enter dwellings

**Genus Bertulia**

*B. validiana* under the bark of trees in Chili

**Genus Leptocimex** Roubaud 1913

*L. boneti* synonym *Cimex bone* Brumpt 1901 found on man in the higher regions of the Ivory Coast and in Haute Guinée

## FAMILY ANTHOCORIDÆ

*Cyrtocera* with embolus membrane with one to four nerves which arise at the tip and along the side of a triangular basal cell Antennæ cylindrical

**Genera**.—*Anthocoris* Fallén 1829 and *Lycocoris* Hahn 1835

*Smell*—The peculiar odour is due to the secretion of sac like

*Prophylaxis*—Bugs are by no means without their enemies of which the most marked is the common red house ant (*Monomorium pharaonis*) while another enemy is the common cockroach but despite these foes bugs are extremely common in tropical hospitals

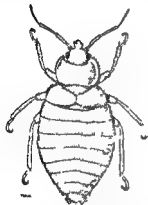


FIG 35—*Clinocoris rotundatus*  
SIGNORNY MALE (X10)

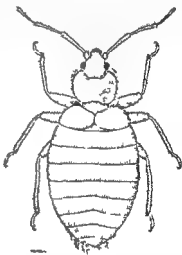


FIG 36—*Clinocoris rotundatus*  
SIGNORNY FEMALE (X10)

The first step in prophylaxis is of course strict cleanliness and the use of painted iron bedsteads which can be easily taken to pieces and washed. Another good means is to make use of mattresses and pillows covered with Willesden canvas which can easily be washed. The mattress should be in sections and not all in one piece as otherwise it is difficult to manipulate. The most useful substances for killing bugs are acetic acid camphor and carbolic acid. Acetic acid poured into the joints of a bedstead is said to kill the bugs rapidly. Kerosene is often used but does not appear to be very effective. Pyrethrum is also used. Apart from bedsteads the whole room may be infected in which case fumigation with sulphur is quite the best remedy, 4 ounces being burnt for each 1000 cubic feet of space and the room left closed for four or five hours in order that the gas may act thoroughly.

very severe and causes much swelling and irritation. It is a night fier and has an odour like that produced by bugs.

**Morphology**—It is a large dark brown insect with pink markings and a flattened body and very narrow pointed head with a strong thick long proboscis. The thorax is provided with wings.

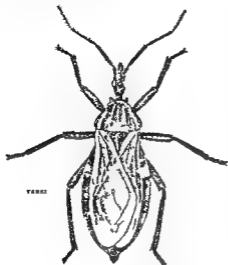


FIG 377—*Conorhinus sanguisugus*  
LECONTE 1855 FEMALE (X2)

**Life-History**—The eggs when first laid are white but later change to yellow and finally to pink. The larva hatches in about twenty days and is very active. It moults twice and becomes the pupa which also moults twice the last stage showing wing pads. The pupa is also active.

**Habits**.—In addition to feeding upon blood these insects may suck the juices of decomposing flesh.

**Pathogenicity**—Its bite at times is followed by severe general symptoms such as swelling in different parts of the body, nausea etc. as well as local pain.

**Remedy**.—Sweet oil is advised as a remedy for the local pain.

*Conorhinus rubrofuscus* Latr. de Geer. This is the Malay bug found in Africa (Sierra Leone and Madagascar) in Asia (Ceylon, India, Malaya, China and the Philippine Islands). It is said to produce a very severe bite.

*Conorhinus venustus* Herrich-Schaeffer is the black bug of the Pampas. *Conorhinus nigrovarius* is the bichugue of South America and bites severely. *Conorhinus protractus* is the big bed bug of Utah.

### Lanus Stal 1859

*Reduviidae* with the head much shorter than the thorax, with a conical preocular portion with the basal segment of the rostrum longer than the apical. Antennae which are inserted a little in front of the eyes are more than twice as long as the head. Ocelli present. Scutellum unarmed, legs rather slender, anterior femora slightly thinner than the posterior and armed with spines.

### *Lanus megistus* Burmeister

#### Synonymy

Chagas

*Trypanoso*

owing to

indigenous population

**Morphology**.—*L. megistus* Burm. is a large black insect with numerous regularly arranged red markings and differs from *C. rubrofuscus* de Geer which is closely allied to it by the fact that

**Anthocoris** Fallén 1829

Two species of importance—*A. kingi* Brumpt 1910 in the Egyptian Sudan and *A. songolensis* Brumpt 1910 in the Belgian Congo

**Lycocoris** Hahn 1835

*Lycocoris campestris* Fabricius is said to attack man

## FAMILY REDUVIIDÆ

*Gymnocerata* with long narrow heads and distinct neck. Eyes large and prominent proboscis short thick and curved antennæ long slender at the tip legs long elytra with three divisions when

*Reduvius Conorhinus*  
but *Harpactor Eulyes*

In addition it may be mentioned that Wellman found *Phonergates bi-*

**Reduvius** Fabricius 1803

pubescent legs very long and slender anterior femora normal anterior tibiae fringed

**Reduvius personatus** Linnaeus 1758

This is the wheel or masked bug found in Europe and the United States and known for the severity of its bite for it causes pain swelling and irritation in the affected area which may last as long as a week

**Conorhinus** Laporte 1837

Synonym—*Tysatoma* Wolf 1802

*Red. d. h. d.*

apex of abdomen placed very far apart prosternum broadly sulcated abdomen frequently with the disc flattened

604

**Conorhinus sanguisugus** Leconte 1855

This is the blood sucking cone nose of America which feeds upon the blood of insects including bed bugs and of mammals including man. The bite is



## FAMILY ARADIDÆ

Broad very flat bugs with four segments in the antenna and three proboscis. No cuneus. Tarsus two segments.

*Dissodius luratus* Fabricius is the Pito bug of South American which bites severely.

## REFERENCES.

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- CHACAS (1909) Bulletin de la Société de Pathologie Exotique  
 DARWIN (1888) Voyage of the *Beagle* p 330  
 DARWIN (1898) U S Department of Agriculture B Entomology Bull (New Series)  
 DARWIN (1900) U S Department of Agriculture B Entomology L No 22  
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*C. rubrofasciatus* is of a dull dark brown colour with markings on the pronotum and with dusky yellow or brick red elytra and connexivum.

**Pathogenicity.**—It is the cause of South American trypano somiasis.

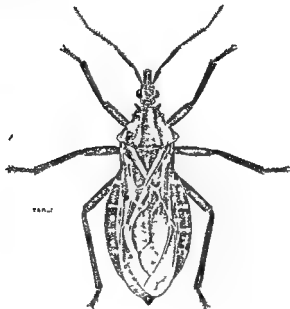


FIG 378—*Laimus megistus* BERNEISEN IEMAI (A.)

**Reduviolus Kirby 183**

*Reduviolus subcolophratus* Kirby 183 has once been recorded as a human blood sucker in the United States.

**Rasahus Amyot and Serville 343**

*Rasahus biquittatus* Say 1831 is found in the houses in Cuba Panama and Para where it really seeks the bed bug but bites man also.

**Melanolestes Stal 1866**

*Melanolestes morso* Erichson 1848 Found under stores during the day in Guiana Mexico and the United States of America Bites man.

*M. abdominalis* Herrich Schaeffer 1818 in the same regions as *M. morio*.

**FAMILY HYDROMLITRIDE**

Up to the present of tropical development of a *Critidia* has been described in the

clature which will be more fully explained under the heading Cubitus but which may here be compared with the typical arrangement of Comstock and Needham from which it differs mainly by the form of coalescence called inward which means that two veins have coalesced from the tip towards the base of the wing.

Typical Names.		Dipteral Names.
Costa		Costa
Subcosta		Subcosta
Radius 1		First Longitudinal
Radius 2	} Coalesced	Second Longitudinal
Radius 3		
Radius 4	} Coalesced	Third Longitudinal
Radius 5		
Media 1	} Coalesced more or less	} Fourth Longitudinal
Media 2		
Media 3		
Media 4		
Cubitus 1		Fifth Longitudinal
Cubitus 2		
Anal 1		Sixth Longitudinal
Anal 2		Seventh Longitudinal
Anal 3		

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## CHAPTER XXVII

### THE DIPTERA

#### CULICIDÆ AND THEIR ALLIED FAMILIES

Diptera . Morphology—Classification—Culicidæ—Corethridæ—Chironomidæ  
—Psychodidæ—Simuliidæ—References

#### DIPTERA

**Definition**—Hexapoda with two well developed transparent wings and two rudimentary wings in the form of halteres. Mouth parts well developed adapted for piercing and sucking or for suction. Mesonotum forms by far the larger portion of the thorax. Metamorphosis is complete.

**Remarks** The Diptera are by far the most important order of the Hexapoda as regards tropical medicine for they include the

nose and cause disease by gnawing away mucous membrane cartilage and even bone of the alimentary canal in which they may

**Morphology**—The most important points in the morphology of the Diptera in general may be briefly mentioned.

*Head*—The head in certain families shows an anterior depressed area

carries an arista on the third joint which may be looked upon as representing the remainder of the larger antennæ of other species.

t blood

may be evolved during the next few years, as there are already signs that such a work -

As the *Anopheles* is, without doubt, of the greatest importance to medical men, its anatomy will be described.

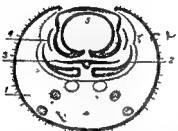


FIG 380 - TRANSVERSE SECTION OF THE PROBOSCIS OF *Anopheles maculipennis* MEIGEN

(After Nuttall and Shipley from the *Journal of Hygiene*)

1, Labium, 2 maxilla 3 hypopharynx with salivary duct 4 mandible, 5 labrum epipharynx with the figure (5) placed in the blood tube

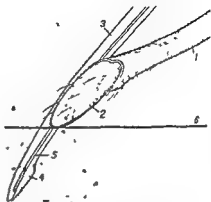


FIG 381 - DISTAL END OF THE PROBOSCIS OF *Anopheles maculipennis* MEIGEN

1, Labium, 2, labellæ, 3 labrum epipharynx, 4 maxillæ 5 mandibles, 6, skin line (The labellæ should be divergent to the plane of the paper)

fifteenth segment bears fine hairs. These few hairs produce quite a different appearance in the antennæ, which are called 'pulose.'

In front of the frons a sclerite called the 'clypeus' belonging to the exo-

black; h but some of the Culicidæ—e.g. *Megarrhina*—are brilliantly coloured. The colours depend partly upon pigments and partly upon interference with or reflection of light.

**Life-History**—The female generally lays eggs more rarely a larva is produced directly as in *Glossina*. The eggs are laid in some material which will be useful to the larvæ—e.g. the eggs of *C. ...*

such a pupa as seen in the Muscidae belongs to the coarctate type—or it may not be so enclosed but the body and appendages being closely united it forms the obtectate type. The imago escapes from its pupal skin by a T-shaped slit (*Orthorrhapha*) or by a circular opening (*Cyclorrhapha*).

*Collectors of Flies*—Mr Austen has asked us to invite the reader's attention to certain remarks of his taken from the second report of the Wellcome

**Classification**—The Diptera may be classified as follows according to the characters of the pupa, larva and antennæ—

### SUBORDER I ORTHORRHAPHA

**Definition**—Diptera without lunule or pitulum. Larva with a distinct head. Pupa obtectate. Imago escapes by T-shaped opening.

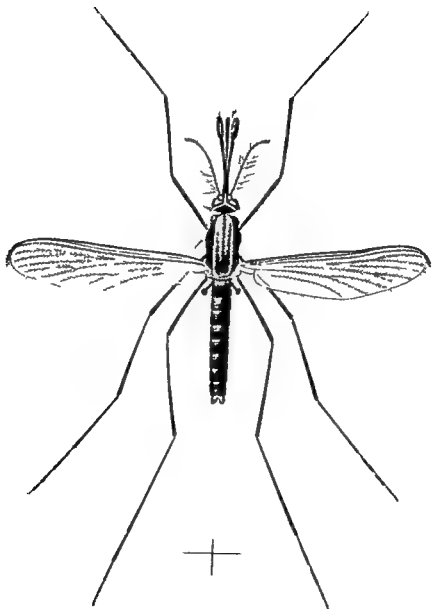
**Section 1 Nematocera**—*Orthorrhapha* with antennæ composed of more than six joints with the joints except the first two similar without arista. Palpi four or five jointed.

**Families**—Culicidæ Corethridæ Chronomidæ Psychodidæ Simuliidæ Blepharoceridæ etc.

**Section 2 Brachycera**—*Orthorrhapha* with antennæ in which the joints differ from one another with or without arista which when present is usually terminal.

**TRIBE I *Brachycera Iomæodactyla***—*Orthorrhapha* brachycera with three well developed pulvilli. Larva with a projecting posterior stigma.

**Families**—Tabanidæ Ieptidæ



ANOPHELES MACULIPENNIS WFIGEN

VALE





and

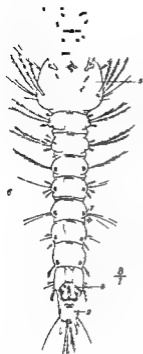


FIG 188.—LARVA OF  
*Anopleis maculipennis*  
(After Nuttall and Shipley  
*Journal of Hygiene*)

1 internal clypeal hairs  
2 external clypeal hairs  
3 antennæ 4 head markings  
5 thorax 6 abdomen  
7 palmate hair ■ stigmatic siphon 9 last segment

the larva escaping from the blunt anterior end of the egg by the shifting off of a piece like a cap from the rest of the shell is seen to consist of head neck and abdomen

When first hatched the head is very black but later on it becomes lighter in colour and shows characteristic markings. At the back of the head there is a little notch and from this a V shaped dark line opens forwards formed by two diverging bands of chitin arranged along which are patches of pigment which give rise to the characteristic markings. There are two large compound eyes behind each of which lies a single eye spot or ocellus.

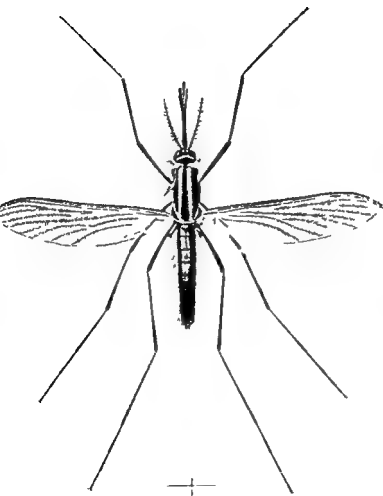
The antennæ are conspicuous rod like bodies ending in two leaf shaped appendages between which is a branched hair while another hair of specific importance arises from a papilla situated at the junction of the proximal and middle thirds.

middle line external to these lie the external clypeal hairs which arise from the outer angle of the clypeus and behind them lie the posterior clypeal hairs. Sometimes there is a basal hair external to the antennæ.

The mouth parts consist of two large feeding brushes two maxillary palps two mandibles and on the ventral median line the under lip of Meinert a conical chitinous structure and a snout like process covered with hairs projecting between the brushes.

The thorax is large increasing in size as the larva grows older. It has numerous hairs and sometimes a pair of the palmate hairs presently to be described.

PLATE III.



ANOPHELES MACULIPENNIS MEIGEN

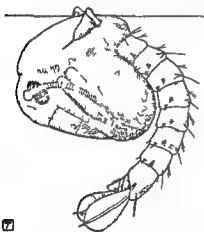
FEMALE.

(After Austen, by kind permission of the Trustees of the British Museum.)

eleven days in *Myomyia rossi* while it may be eighteen to twenty one days in *Anopheles maculipennis* in the temperate zone. The larva grows by moulting several times.

Culicine larvæ are easily distinguished from Anopheline larvæ by the fact that the spiracles are carried on a long respiratory siphon formed by a prolongation of the dorsum of the eighth abdominal segment which has been used to distinguish the different species (Fig 390).

Below the siphon on the eighth segment there are spines forming a comb while along the length of the siphon there is another comb distal to which is a tuft of hairs. The variations in these structures together with those in the length and breadth of the siphon associated with those of the antennæ and clypeus afford means of classifying the Culicine larvæ. For further information consult either



7

FIG 391 — PUPA OF *Anopheles maculipennis*

(After Nuttall and Shipley *Journal of Hygiene*)

Theobald's Monograph vol 11 p 6 or Felt's paper Bulletin 97 of the Division of Entomology of the New York State Museum. The Megarhininæ, Ædinæ and Uranotæninæ possess larvæ belonging to the Culicine type.

The pupal stage lasts about forty eight hours. Towards the end of an afternoon the pupa comes up to the surface and the dorsal portion of the thorax splits with a T shaped fissure and the adult insect or imago emerges. Pupæ do not eat.

Differences between the Anophelinæ and Culicinæ — The difference between these two important families may popularly be described as follows:

The Anophelinæ as a rule project from any plane surface on

which they may be resting at a sharply defined angle owing to their head, thorax and abdomen forming a more or less straight line while the Culicinæ on the other hand do not make such a well defined angle owing to the fact that the abdomen is not in

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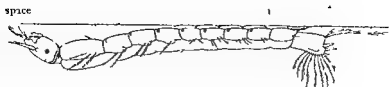


FIG 389—LARVA OF AN ANOPHELINE MOSQUITO LYING PARALLEL TO THE WATER  
(Modified after Howard)

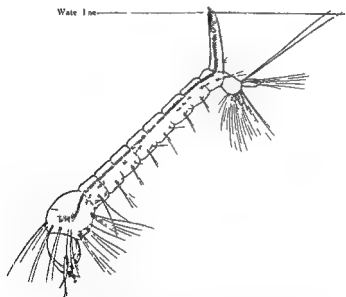


FIG 390—LARVA OF A CULICINE MOSQUITO HANGING DOWN FROM THE SURFACE OF THE WATER  
(After Howard)

The teeth are of great importance being capable of being approximated so as to close the cavity and thus protect the two openings of the tracheæ which lie in the anterior portion of the siphon. From these openings the long tracheæ can be seen running from back to front along the larva.

The duration of the larval stage varies with food and temperature being longer in the temperate zone than in the tropics. According to Stephens it is twelve days in *Culis argyrotarsis* and

In the early morning the female flies to the nearest water and lays her eggs. Usually she does not travel far but is believed to be capable of going at least half a mile in case of need.

The females of the *Culicine* do not appear to concern themselves as to the nature of the water in which they lay their eggs but the female *Anopheline* prefer clean water with a certain amount of weed. This water may be the back eddies of a river where there is the protection of weeds or the margins of large lakes where there is a pool or any collection of water or furling these may collect

Mosquitoes can hibernate during the cold weather of the temperate zone and aestivate during the dry hot weather of the tropics.



FIG. 396—*Girardinus pacilloides* DE FILIPPI  
(The tail should have been drawn expanded)

In this latter condition they bite and suck blood but apparently do not lay eggs even if water is provided. The eggs are kept afloat on the surface of the water by their structure and in due course give rise to the larvæ which are great eaters living not merely upon unicellular organisms such as algæ and diatoms but also upon their fellows. For purposes of obtaining air they are compelled to come to the surface of the water. The *Anopheline* larvæ not possessing a siphon has to lie more or less parallel with the

apex of the siphon to the surface of the water from which they apparently hang downwards. Larvæ are certainly able to hibernate and perhaps eggs also. The pupa does not feed.

Mosquitoes have many enemies and parasites but the most important from a point of view of the prophylaxis of malaria are those which eat the eggs, the larvæ and the pupa, of which fish are the most important.

In 1905 C. K. Gibbons pointed out that a small fish popularly

upon vegetable juices, though this is more common in the females of the Culicidæ than in those of the Anophelina. It is believed



FIG 392—DIAGRAM TO SHOW THE POSTURE OF AN ANOPHELINE MOSQUITO ON A WALL

(After Sambon from the *British Medical Journal*)



FIG 393—DIAGRAM TO SHOW THE POSTURE OF ANOTHER ANOPHELINE MOSQUITO ON A WALL



FIG 394—DIAGRAM TO SHOW THE POSTURE OF *Culex pipiens* ON A WALL

(After Sambon from the *British Medical Journal*)

that a female feeds on blood once a day in nature, but this is a difficult matter to be certain about. The mechanism of the bite has already been described in page 223, to which reference should be made. It will also be noted that the structure of the female mouth-parts is adapted for piercing, while that of the male is not. It will also be remembered that only the stylets pierce the skin, and that the labium never does so. Infection of the victim by the malarial germ takes place during the act of



FIG 395—RAFT OF CULICINE EGGS  
(After Sambon)

biting as the sporozoites pass down the hypopharyngeal or salivary tube, while the infection of the mosquito is effected by the blood, which passes from the victim along the labial or blood-tube into the mouth. So

at dinner at night.

After feeding, the mosquitoes usually retire to a dark portion of the room to digest the food. It is noticeable that they avoid white areas during the daytime, and prefer dark coloured regions away from the light, and hence are very difficult to find in ill-lighted native huts.

which Theobald has modified and brought into accord with one based upon scales as generic characters and this classification is followed below

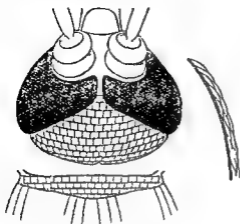


FIG. 397.—HEAD AND SCUTELLUM OF MEGARHINUS TO SHOW THE SCALES

On the right a profile view of the scales  
(After Theobald Culicidæ of the World)

**THEOBALD'S CLASSIFICATION**—The Culicidæ may be divided into subfamilies according to the characters of the scales on the head body legs and wings

A Scutellum simple never trilobed proboscis straight palpi long in male and female—*Anophelinae*

B Scutellum trilobed—

I Proboscis strongly recurved first submarginal cell very small—*Megarhininae*

II Proboscis straight post scutellum nude—

I Wings with six longitudinal scaled veins—

(1) Antennæ with second joint normal in length—

(a) First submarginal cell as long as or longer than the second posterior cell

Palpi in the female shorter than the male

Palpi in the female shorter than the male

Palpi in the female shorter than the male

(2) Antennæ with second joint very long—*Deoceratinae*

II Wings with seven longitudinal scaled veins—*Heptaplebomyinae*

III Proboscis straight post scutellum with scales or chaetae—

1 Palpi long in male short in female—*Trichoprosopinae*

2 Palpi short in both sexes—*Dendromyinae*

IV Proboscis elbowed—*Limatinae*

Of these only the Anophelinae and the Culicinae and possibly the Aedinae contain species of importance in tropical medicine and concerning these subfamilies a few details must be given

termed millions which lived in shallow water was a voracious

is distinguished by red splashes and a black circular dot on its sides. The great importance of these little fish is that they are able to live in very shallow water and to work their way in among dense surface vegetation and thus to gain access to the larvæ etc. of the mosquitoes which otherwise are protected by the weeds from attacks by the larger fish. Their classification is Teleostei Haplomi Cyprinodontide genus *Haplochilus* McClelland 1839. The family contains 220 species of which only 50 live out of America. *Haplochilus* has 24 species in Africa and 35 in Asia and America. There is no doubt that these small fish should be introduced into malarious places as a prophylactic measure against the disease. Other closely allied species are *G. versicolor* Gunther found in St. Domingo and *G. formosus* found in Florida and South Carolina. With regard to other species *Gambusia molliensis* is said to be of great value in consuming larvæ especially when protected by dense surface vegetation. Recently Graham has reported that *Haplochilus grahami* Boulenger 1911 and *H. bifasciatus* Steindachner 1881 of the Cyprinodontide eat larvæ greedily while Gowdey finds the same for *Fundulus tenuis pignus* and *Haplochilus pumilus* Boulenger 1906 in Uganda.

Certain plants as is well known collect water especially the bromelias the bamboos and the pitcher plants. In this water Culicine and sometimes Anopheline larvæ can be found. E. E. Green of Ceylon has shown that the flowers of the lobster claw plant (*Heliconia brasiliensis*) can hold a considerable amount of water in which he found *Stegomyia* and *Desmoulinia* larvæ in large numbers.

**Classification**—Various methods of classifying mosquitoes have been brought forward. The early ones were based upon the characters of the palpi but in 1901 Theobald showed that they were useless for anything but specific characteristic and based his larger divisions on the variations of the scales on the head, body, and wings. He brought forward a classification based upon the male genitalia and the wing veins but as Theobald remarks the majority of known mosquitoes being female it is most undesirable to take only male characters for the classification. Recently Dyar and Knab have issued a classification of the Culicidæ by larval characters and Fyell has advanced matters considerably by separating the Corethridæ from the Culicidæ under the term Corethrida because the Corethridæ have an entire absence of the long piercing proboscis and of scales in the adults both of which are marked features of the Culicidæ. It will be evident that this is useful. Lutz has brought forward a classification based upon larval and adult forms



- 3 Thorax with hair like curved scales and some narrow curved ones in front abdomen with apical lateral scale tufts and scaly venter no ventral tuft Wing scales lanceolate—*Arribal* sp: Theobald
- 4 "
- 5
- 6 Thorax with very long hair like curved scales abdomen with hairs except last two segments which are scaly Dense scale tufts to hind femora Wings with broadish blunt lanceolate scales—*Lophoscelomyia* Theobald
- 7 Thorax and abdomen with scales —
- (a) Thoracic scales narrow curved or spindle shaped abdominal scales as lateral tufts and small dorsal patches of flat scales—*Yssorhynchus* Blanchard
- (b) Abdomen nearly completely scaled with long irregular scales and with lateral scale tufts—*Celisa* Theobald
- (c) Similar to above but no lateral scale tufts—*Acrotelia* Theobald
- (d) Abdomen completely scaled with large flat scales as in *Culex*—*Aldrichinella* Theobald
- (e) Thoracic scales hair like except a few narrow curved ones in front abdominal scales long broad and irregular—*Heritszia* Theobald
- (f) Thorax with narrow hair like curved scales some broad straight scales and some spatulate laterally Abdomen with fine hairs except last three segments which have scales Tufts of scales on hind femora Wing scales lanceolate—*Manguinostia* Cruz

NOTE—The genus *Calodiazess* Dyar and Knab is said by Theobald to be invalid being based on *Anopheles barberi* which is a true *Anopheles*

#### *Anopheles* Meigen 1818

Essentially *Anopheles* are temperate zone or hill station Anophelinae of which the type *Anopheles maculipennis* Meigen 1818 has been already described

#### ANOPHELES

##### 1 Wings spotted —

##### 1 Legs unbanded —

1 Wings with spots formed of collections of scales on the wing field no costal spots—*maculipennis* Meigen

2 Wings with light and dark costal markings

(1) Costa with two yellow spots —

(a) Large species —

(A) No fringe spots—*punctipennis* Say

(B) Fringe spots present—*pseudopunctipennis* Theobald

(b) Small species Wings much spotted—*franciscanus* McCracken

(2) Costa with one spot—*perplexus* Ludlow



- 3 Legs unbanded —  
 (a) Apex of palpi black — — — — — 4 bands  
 (A)  
 (B)
- (3) No fringe spots—*rhodesiensis* Theobald  
 (b) Apex of palpi white only—*nsis* Theobald  
 (c) Apex of palpi black  
 (1) Black apex narrow—*irrhuda* Liston  
 (2) Black apex broad—*hispaniola* Theobald
- 4 Legs with spots only at joints Palpi with three bands apex black  
 —*arshi* Patton
- II Proboscis banded —  
 I Legs unspotted—*albirostris* Theobald  
 II Legs spotted—*thorntoni* Ludlow

### *Neomyzomyia* Theobald 1912

This genus includes only one species *Neomyzomyia elegans* James 1903 It is found in India

### *Pyretophorus* Blanchard 1902

The important species is *Pyretophorus costalis* the spreader of malaria in West Africa and Mauritius

### PYRETOPHORUS

- A Legs unbanded —  
 I Palpi with three pale bands apex black—*nigrifasciatus* Theobald  
 II Palpi with three pale bands apex white —  
 (a) Wings with four large and two small black costal spots mid cross vein very long—*nurses* Theobald  
 (b) Wings with four large black spots mid cross vein normal—*minus* Theobald  
 (c) " — " — — — — — the second posterior—  
 the second cell—*palast*  
*nurses* Theobald
- B Legs banded —  
 I Legs with apical banding Hind legs only banded Palpi black  
 apex and three pale bands — — — — — id
- I  
 (b) Wings with four large and two small costal spots fringe spotted  
 (A) Apical palpal band broad other two small—*jeyporensis*  
 Theobald  
 (B) Apical and median palpal bands broad—*austeni* Theobald  
 (c) Thoracic scales creamy—*pitchfordi* Power
- III Fore and hind legs with apical pale bands  
 Four white palpal bands—*cinerens* Theobald
- C Legs spotted and banded —  
 I  
 Theobald  
 II  
 bands apical Three palpal  
 bands

bald

ei Theob

B Wings unspotted —

I

(1) Palpi unbanded

(A) Petiole of first fork cell more than one third length of cell

A L 2      Ab      T 2      L      L 2      T 2      T 2

(B)

(2) Palpi banded Dark species Wing scales very dense  
—*smithi* TheobaldWing scales not so dense—*nigripes* Staeger(b) Second fork cell not more than half the length of the first  
—*asthani* Theobald

II Legs banded —

1 Hind femora only with broad white band—*lindsaysi* Giles2 Apices of hind tarsi pale—*immaculatus* Theobald

## Myzomyia Blanchard 1902

This *Myzomyia* includes some important mosquitoes found in West Africa and in India and Ceylon which are carriers of malaria

The diagnostic table given by Theobald is as follows —

## MYZOMYIA

A Proboscis unbanded —

I Legs banded.—

(a) Palpi with three white rings

(1) Legs with faint apical pale bands

Wing fringe spotted—*funesta* Giles(2) Legs with prominent apical pale bands and a broad pale median band to fore and mid metatarsi—*fulva* Theobald

(3) Legs (hind) with apical and basal pale band

Wings with five to six pale costal spots the largest T-shaped—*ros* Giles(c) Supernumerary cross vein markedly curved—*pyrelophoroides* Theobald

- (g) Thorax with white frontal median spot two large lateral spots one small spot on front of wings one narrow median white line and narrow submedian lines on posterior half Last two hind tarsi white—*wellmani* Theobald
- (h) Thorax brown with broad white line in front extending laterally towards the wings where they swell into a large patch a white line just behind wings Last two hind tarsi white—*albipes* Theobald
- (i) Thorax with silvery white spot on each side in front small spot over root of wings and a white spot over the base of the wings—*pseudonigera* Theobald
- (j) Thorax with two lateral white spots the front one the largest a small median one near the head two yellow median lines and a short silvery one on each side before the scutellum—*simpsoni* Theobald
- (k) Thorax with a silvery white scaled area in front and another on each side in front of wings—*argenteomaculata* Theobald
- (l) Thorax with a median yellowish white line a silvery patch on each side in front of the wings extending as a fine yellow line to scutellum and another silvery spot before base of each wing—*powellii* Theobald
- (m) Thorax with small grey scaled area in front of roots of wings and three short creamy lines behind—*minutissima* Theobald
- (n) Abdomen black fifth segment with yellow basal band sixth unbanded (seventh) two medio-lateral white spots (eighth) two baso-lateral white spots second hind tarsus nearly white—*dubia* Theobald
- (b) Abdomen unbanded
- (1) Third hind tarsal nearly all white  
Thorax with two lateral white marks directed upwards—*africana* Theobald
- (2) First hind tarsal all white  
Thorax with one white spot anteriorly and one in front of each wing—*apicoargentea* Theobald  
Thorax chestnut brown—*terrens* Walker
- II Legs with white lines as well as basal bands  
Thorax brown with white lines abdomen with basal bands—*grahni* Theobald
- III Fore and mid legs with apical bands hind basal  
Fourth tarsal of hind legs nearly all white — *mediopunctata* Theobald  
Base of mid metatarsi base and apex of hind and base of first tarsal with pale banding—*assamensis* Theobald
- IV Legs unbanded.—
- (a) Abdomen basally banded
- (1) Thorax with front half silvery white remainder bronzy brown—*pseudonivea* Theobald
- (2) Thorax deep brown with scattered golden scales—*alboccephala* Theobald
- (b) —
- (c) Abdomen unbanded  
Thorax with six silvery spots—*argenteopunctata* Theobald
- (d) Abdomen with apical white lateral spots  
Thorax unadorned except for pale scaled lines laterally—*punctolateralis* Theobald

{A} Apical one broad others narrow —

(b)  
(c)

d

apical

### Myzorhynchus Blanchard 1902

These mosquitoes are usually said to only occur in the open but we have repeatedly found *Myzorhynchus barbrosus* in houses. *M. sinensis* is known to carry the parasite of malaria in Japan. No species have to far been

### MYZORHYNCHUS

A Palpi unbanded —

I Last hind tarsals brown Legs with pale apical tarsal bands —  
(a)

Ludlow

(b) Several fringe-spots—*lancofti* Giles

(c) No fringe spot

(1) One pale costal spot wings with light and dark scales—  
*umbrosus* Theobald

(2) Two pale costal spots wings mostly dark scaled—*strachani*  
Theobald

II Last hind tarsal white—*albatrossatus* Theobald

B Palpi banded —

I

sedemann

as Walker

spots—*pseudofictus*

2 Wings with two white costal spots—*sinensis* Theobald

(b) Apex of palpi black—*nigerrimus* Giles

II Last two hind tarsals white—*aurisianus* Grandpré

III Last three hind tarsals white—*paludis* Theobald

### Nyssorhynchus Blanchard 1902

The important member is *Nyssorhynchus fuliginosus* Giles 1900 which is without doubt a malarial carrier. Theobald's diagnostic table is as follows —

### NYSSORHYNCHUS

A Last hind tarsals brown —

Legs spotted —

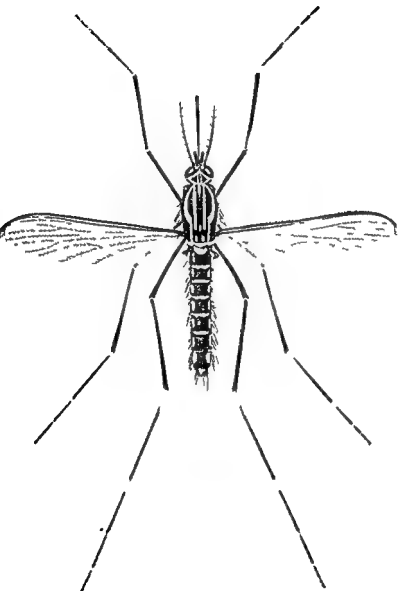
(a) Apical pale bands to legs

(1) Proboscis dark—*stephensi* Linton

(2) Proboscis pale on apical half—*mastersi* Skuse

(b) Apical and basal pale banding—*annulipes* Walker





STEGOMYIA CALOPUS MEIGEN  
FEMALE



scaled longitudinal veins and with the first submarginal cell as long as or longer than the second posterior Post scutellum nude

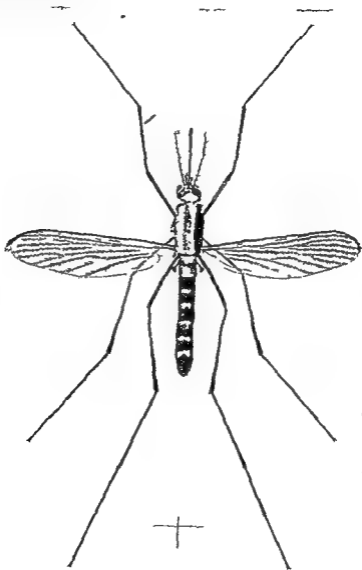


FIG 400—*Culex pipiens* LINNÆ 5

The mosquitoes included in this subfamily are generally found in the jungle and not in habitations. De Meillon thinks that perhaps they may be proved to be malarial carriers (see

# GULEX

- (e) Abdomen with basal white lateral spots  
 (1) Thorax with two pale indistinct median parallel lines and two silvery lateral spots—*minuta* Theobald  
 (2) Thorax unadorned  
 White spot mid head—*tripunctata* Theobald  
 No white spot—*amesii* Ludlow

Proboscis yellow basally dark apically  
 Abdomen with apical pale bands—*crassipes* van der Wulp  
 Proboscis with median interrupted white line on basal half  
 Head black with grey margin—*albomarginata* Newstead

NOTE—*S. lambersi* Ventbrillon *S. leucomeres* *S. desmotes* Giles *S. stracocura*  
 Miles of uncertain position

*Culex* Linnaeus 1758

**Definition.**—Culicids with head covered with narrow curved scales above flat scales at the sides and upright forked scales Male palpi long acuminate female short Thorax with hair like curved scales or narrow curved scales Linear lateral vein

**Remarks.**—The important species are *Culex pipiens* Linnaeus and *C. fatigans* Wiedemann

*Culex pipiens* Linnaeus 1758

**Synonyms.**—*Culex vulgaris* Linnaeus 1767 *C. alpinus* Linnaeus 1767 *C. agilis* Bigot *C. ciliaris* Linnaeus 1767 *C. communis* de Geer, *C. rufus* Meigen 1818 *C. phytophagus* Ficalbi 1889

*C. pipiens* is widely distributed in Europe North Africa and North America where it is the common brown mosquito

Its general appearance is well shown in the illustration from which the head is brownish from the golden brown scales as are the proboscis palpi and antennae The thorax is dark brown with black lines due to black bristles Legs are brown and unbanded

*Culex fatigans* Wiedemann 1828

*C. fatigans* Wiedemann 1828 *C. pungens* Wiedemann 1829  
*C. fallipes* Meigen 1838 *Helicomyia dolosa* Urbalzagala 1896  
 This is the common brown house-mosquito of the tropics which is believed to spread dengue fever  
 It resembles the above but has only two dark lines on the thorax as well as the basal abdominal bands are white or pale cream colour and the fork cell is longer

*AEDINA* Theobald

**Definition.**—Culicids with straight proboscis short palpi in both sexes usually plumose antennae in the male pale in the female Wings

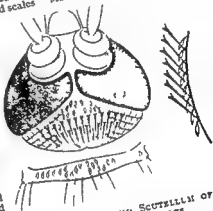


FIG. 309 HEAD AND SCUTELLUM OF *CULEX* TO SHOW SCALES  
 (After Theobald) Culicids of the World

golden brown curved scales and with the  
 Abdomen brown with basal yellow bands

## SUBFAMILY CERATOPOGONINÆ Kieffer, 1899.

**Definition**—Chironomidæ with the thorax not prolonged over the head, antennæ with fourteen rarely thirteen, joints in both sexes and the last joint never longer than the two preceding taken together, while the last five are longer or almost the same as the preceding joint

**Life-History**—The eggs may be laid on land or water. If they are laid on land the larvæ and pupæ resemble those of *Phlebotomus* presently to be described, while if they are laid in water they occur in small clusters of thirty to sixty eggs. The larva is snake-like and transparent, and lives on the surface of stagnant water or nally develops athing organs

**Remarks**—The subfamily has been especially studied by J J Kieffer who recognizes the following genera *Leptoconops* Skuse

say as the descriptions are too imperfect

## Key of Genera

- (a) Wings absent or rudimentary
- (aa) Wings present
  - (f) wings hyaline
  - (gg) Wings spotted—(1) *Ecacia*
  - (ff) Antennæ fourteen or less joints
    - (g) Thorax rounded and not produced over the head. Antennæ thirteen to fourteen joints legs of moderate length
      - (h) Antennæ thirteen joints—(2) *Leptoconops*
      - (hh) Antennæ fourteen joints plumose in the male sparsely paired in female. Typical wing venation
    - (i) Wings hairy last joint of tarsus with an empodium
      - (j) Empodium well developed almost as long as the claws which are without setæ—(3) *Ceratopogon*
      - (jj) Empodium not so distinct less than half as long as the claws which have setæ on the underside—(4) *Culicoides*

— of them (*Hantagopus* ?) as possible

the respiratory siphons. A  
 the work should be consulted

### LIMATINÆ Theobald.

**Definition.**—Culicidæ with elbow bent proboscis and squamæ on post scutellum. Palpi short in both sexes. First fork cell longer than second.

**Genus.**—*Limatus*

### FAMILY CORLTHIRIDÆ

Eysell, 1905

**Definition.**—*Orthorhapha nemocera* with short proboscis not formed for piercing, without scales in the adult condition, with transparent larvæ rather resembling those of *Chironomus*

**Remarks.**—The only reason why this family, which includes the genera *Corethra* and *Mochlonyx*, is mentioned here is that it has only recently been separated from the Culicidæ, of which it forms a subfamily—Culicimorphæ



FIG. 401.—HEAD AND SCUTELLUM OF CULICIDÆ TO SHOW SCALES (After Theobald *Culicidæ of the World*)

### FAMILY CHIRONOMIDÆ

**Synonyms.**—*Tipularia culiformis* Culicites Newmann *Tipulada* Leach, *Chironomus* Zelt, *Chironominae* Rondani

**Definition.**—*Orthorhapha nematocera* with head small often retracted under and covered by the thorax. Ocelli absent. No transverse suture on thorax. Eyes reniform. Antennæ from six to fifteen joints. Pectinate in male simple and composed of fewer joints in female. Wings without veins along the posterior margin, costal vein ending near the tip of the wing.

**Remarks.**—The Chironomidæ include over 800 species of very delicate and often quite minute flies, popularly called 'mudges,' which are found all over the world, especially near water.

Kieffer classifies the family into three subfamilies as follows—

A. Media and cubitus united by a cross vein *Tanypidæ*

B. Media and cubitus united only at the base

- I. Thorax humped over the head—*Chironominae*
- II. Thorax not humped over the head—*Ceratopogoninae*.

Of these three subfamilies only the last concerns us

although Austen states that in the type specimen of the species *C. castaneus* Walker 1848 the abdomen is apparently distended with blood as far as can be judged by external examination

**Culicoides Latreille 1809**

**Synonyms**—*Ceratopogon* Meigen 1803 *pro parte*, *Cheironomus* Fabricius

*non* - - -

with the second to eighth joints cylindrical ovate and the next four or five more elongate and subcylindrical and the last joint ovate and cylindrical Subcostal vein ending much beyond half the length of wing radial ending

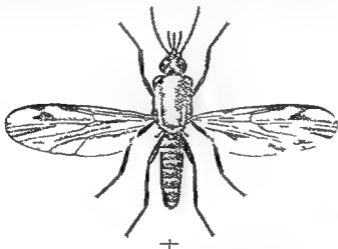


FIG 402—*Culicoides pulicaris* FEMALE  
(After Austen British Blood Sucking Flies )

near tip cubital ending by the tip Abdomen composed of eight segments Legs almost equal in length femora armed beneath with spines

**Type**—*Culicoides pulicaris* Linnæus

There are over one hundred known species of this genus (and many more have been described since this statement was first

they do most  
disease still  
very small,  
The eggs



## Palpomyia Mergele

Synonym.—*Ylocrypta* Kieffer

*Ceratopogoninae* with bare wings and five radial cells  $R_2$  present media simple some or all the femora spinose underneath pulvilli and empodia wanting

Four subgenera *Alasion* Rondani 1857 *Sphaeromyas* Stephens 1829  
*Serromyia* Mergele 1818 *Heteromyia* Say 1825

## FAMILY PSYCHODIDÆ

Definition—*Orthorrhapha nematocera* without ocelli and with body densely covered with coarse hairs Thorax without transverse suture Antennæ long sixteen jointed Wings very broad and hairy No discoidal cell Legs long tibiæ without spurs

Remarks—The members of this family are small sometimes very

*Sycorax* belonging to the *Phlebotominae* and possibly one in the *Psychodinae*

The *Psychodidæ* are classified into two subfamilies—

(1) *Psychodinae*—*Psychodidæ* in which the second longitudinal vein gives off its first branch in the root of the wing The female

cond long  
body of the

wing The female has not got a horny ovipositor while the male has three claspers

## SUBFAMILY PSYCHODINÆ

The genera *Pericoma* and *Psychoda* and their allies belong to this subfamily As a rule it is stated that these flies are not blood suckers but Howlett states that an Indian species occasionally sucks blood

## SUBFAMILY PHLEBOTOMINÆ

This subfamily is of importance not merely because it contains blood sucking flies, but because the genus *Phlebotomus* is accused of spreading the Three Days Fever The two blood sucking genera may be distinguished as follows—

(1) Two simple veins between the forks of the second and fourth longitudinal veins—*Phlebotomus*

(2) One simple vein between the forks of the second and fourth longitudinal veins Seventh longitudinal vein very short—*Sycorax*

*Phlebotomus* Rondani 1840

Morphology—*Phlebotominae* with mouth parts formed for piercing and sucking palpi of five joints antennæ long filiform composed

of the naked species are laid in water attached to floating algae and give rise to white worm like larvæ and small pupæ with prominent respiratory horns. The eggs of the hairy species are laid in decaying vegetal matter and give rise to small larvæ.

### *Culicoides grahami* Austen 1909

**Synonyms**—*Culicoides habereri* Becker 1909 *Cæcia hostilissima* Pittaluga 1910

This minute fly appears to be extremely common and to be almost the most troublesome of these blood thirsty insects in tropical Africa. It is known in the Spanish Guinea Ashanti Congo Free State Uganda Kamerun Southern Nigeria Angola. For fuller particulars see Austen's African Blood Sucking Flies p. 7, Plate I Fig. 3 and Pittaluga's works.

### *Culicoides varius* Winnertz 1867

This is the blood thirsty species in Europe.

### *Cæcia* Poey 1851

This genus is closely related to *Ceratopogon* and *Culicoides*. The antennæ have fifteen and the palpi five joints. Ocelli are present. The wings have

81441: Austen 1912

### *Cæcia furens* Poey 1851

This is the jeyen of Cuba which is said to be very irritating. Its length is 2 millimetres from the head to the end of the abdomen. Legs and antennæ rufous. Thorax bronze-coloured with fuscous spots, abdomen fuscous, legs with whitish articulations and a ring upon each femur tibia fuscous. Wings broad whitish with fuscous spots. They are covered with minute scales and possess conspicuous marginal fringes.

### *Bezzia* Kieffer 1899

**Synonym**—*Ceratopogon* Mcigen 1813 *pro parte Ceratopogonina* with bare wings and tarsi without empodia. Radius with three branches.

**Type**—*Bezzia ornata* Mcigen 1803

### *Brachypogon* Kieffer 1899

*Ceratopogonina* with bare wings, media coalescent with  $R_4+$ , p. villi absent.

**Type**—*Brachypogon viscosus* Winnertz 1852 ( ) 1847 (?)

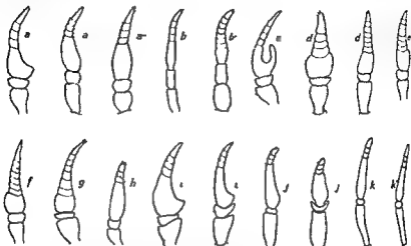
### *Ceratolephus* Kieffer 1899

**Common**

*Ceratopogonina* with bare wings, tarsal claws with 11 teeth. *C. fulcitur* Austen 1912 is the



with well marked labial palps (labella). The wings have a large discal cell and very elongate basal cells. When at rest the wings diverge at the tips. They may be mottled. The legs are large and strong. The body is brown whitish or yellowish in colour but there may be markings on the abdomen.



TABANUS

FIG. 408.—ANTENNÆ OF THE TABANIDÆ

a a a\* *Silvius* b b *Chrysops* c *Rhonomyza* d d *Cadicera* e *Dorca*  
*læmus* f *Pangonia* g *Erephopsis* h *Lepidoselaga* i i *Tabanus* j j  
*Hæmatopota* k k *Hippocentrum*

**Life-History**—The eggs which are spindle shaped and whitish in colour are laid in raft or flask shaped masses attached to water plants. The larvæ live either in water or damp earth and feed upon small animal organisms. They are spindle-shaped and segmented with knobs or protuberances on the rings either all round or only ventrally. The pupa which is free is found in water or damp rubbish.

FIG. 409.—LARVA OF A TABANUS ( $\times 2\frac{1}{2}$ )

at they may disseminate trypanosomes in Timbuctu and spread by *Tabanus dilatatus*. *Trypanosoma* is spread by *T. nemoralis*. Meigen and *T. nigritus* Fabricius. *T. glaucopsis* Meigen is infected with *Herpetomonas subulata*. According to Leiper *Chrysops* is the carrier of *Loa loa* Cobbold 1864.

normally of sixteen segments. The thorax is mainly mesothorax,

means the other simple vein being the third longitudinal. The

inferior claspers, submedian lamellæ and intermediate appendages and a penis.

The buccal cavity is wide in front and narrow behind where it leads via the pharynx into the œsophagus which divides posteriorly

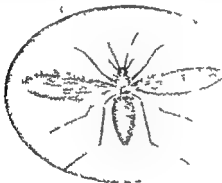


FIG. 403.—*Phlebotomus d. iboscq* n. NEVET LEMAITRE 1906  
(From a photograph by J. J. Bell)

into two tubes, one leading to the sucking, and the other to the mid gut, at the posterior extremity of which are situated the four Malpighian tubules, after which come the small and large intestines. Attention is invited to the presence of the sucking stomach and to the number of the Malpighian tubules.

The female organs consist of ovaries from which the tubular oviducts pass to unite before reaching the base of the inferior claspers. There are two spermatheca.

The male organs are testes, seminal vesicles, ejaculatory duct, pompetta or little pump—which regulates the exit of the spermatozoa—and penis.

**Life-History**—After fertilization the female takes a meal of blood, even though she may have previously sucked blood. She then lays some thirty to eighty eggs in damp places, usually cracks in rocks, stones, or bricks, and in doing so is apparently much

very blood-thirsty, and are a terrible pest in the wet season to man and beast alike. They are said never to bite in the early morning or after sunset, but, on the contrary, Mayer states that *H. decora* is most troublesome in the early morning and late evening. Neave



FIG 411—*Leptoselago lepidota* WIEDEMANN FEMALE (X6)

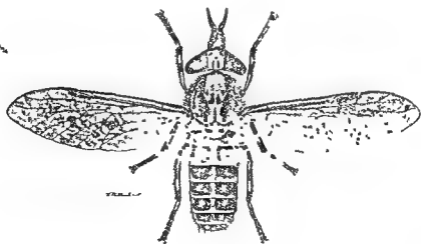


FIG 412—*Hamatopota vittata* LOEW (X4)

states that the females bite in rather dull, damp weather, and rarely in the heat of the sun, being mostly in evidence from about 5 p.m. to sunset in sunny weather, but in

ophylaxis.—Kerosene spread on water appears to be the best  
 rod of dealing with these pests

classification.—The family is divided into two subfamilies  
 aninæ and Paganinæ

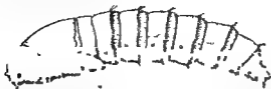


FIG 410.—PUPA OF *Tabanus kingi* AUSTEN (AFTER KING)  
 in the Fourth Report of the Wellcome Tropical Research Laboratories  
 Khartoum)

### TABANINÆ

Tabanidæ without spurs on the hind tibiæ

The important genera of the Tabaninæ may be recognized by the  
 following table —

I Thorax and abdomen with iridescent tomentum—*Lepido-*  
*selaga*

II Thorax and abdomen without iridescent tomentum

I Eyes bare

(a)

(b)

#### *Lepidoselaga* Macquart 1838

Synonym — *Hadrus* Perty

*Lepidoselaga lepidota* Wiedemann 1828 the motuca fly of Brazil,  
 a well known biter (Antennæ vide Fig 408 h)

#### *Hæmatopota* Meigen 1803

... together while the rest of the wings  
 ... backwards and outwards The species of *Hæmatopota* are

## Other Genera

In 1906 Grunberg described three genera with one species in each as

## PANGONINÆ

Tabanidæ with hind tibiae armed at the tips with spurs

## I Proboscis short

1 Antennæ longer than the head—*Chrysops*

2 Antennæ shorter than the head—*Silvius*

## II "

*Adicera*  
the head often

*Chrysops* Meigen 1803

This genus has become of greater importance since Leiper has shown that it includes the carrier of *Loa loa* Cobbold 1864 which

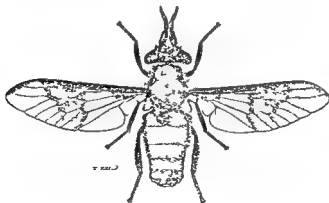


FIG 413—*Chrysops wellmani* AUSTEN FEMALE (X4½)

found in the Ethiopian region The African flies belonging to the

## HIPPOCENTRUM

life history of the tropical species is but little known, but thinks that the majority of the species may have two broods. The life-history of *H. pluvialis* Linnaeus is partially known. The first stage has not so far been recorded. The pupae are of the type common to pathogenic species, probably *H. t.*

... .. Nigeria.

... ..

This genus according to the most recent authorities the antennae extremely slender = first 10 segments =

or bic  
Spei  
17 ...

... .. been  
... .. Africa  
... .. bite man and animals. The life history is unknown.

*Tabanus* Linnaeus, 1761.

There are ... ..

... .. 1909), which is a true parasite.  
*T. hilaris* and another species

A number of  
*micans* Meigen  
is mentioned.

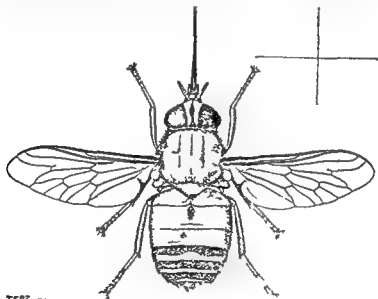
Subgenus *Atylotus* Osten Sacken, 1876.

*Atylotus fulvus* Meigen and *A. rusticus* Fabricius are met with in L.

Wiedemann 1819 *C. chrysostigma* Wiedemann 1818 *C. ribra marginata* Macquart 1885 *C. quinquefasciata* Austen 1912 *C. biclausula* Loew 1860 *C. chrysophila* Macquart 1834 *C. distantis* Austen 1912 *C. speciosa* Austen 1912 *C. flavicornis* Austen 1912 and *C. obscura* Ricardo 1908. The females have been observed to bite man but very little is known as to the habits or life history of the species of this genus.

### *Pangonia* Latreille 1802

This widely distributed genus used to contain a very large number of species but has of late been split up into several subgenera



TERZ —

FIG 415—*Pangonia r. rpell*: JAEVNICKE 1867 FEMALE (X3)

as may be shown in the following table taken from Miss Ricardo's paper in *The Annals and Magazine of Natural History* Series 7 vol 5 January 1900 —

- I Wings with first posterior cell closed
  - (a) Eyes bare—*Pangonia* Latreille (subgenus *Pangonia*)
  - (b) Eyes hairy—*Pangonia* Latreille (subgenus *Ereptopsis* Rondani)
- II Wings with first posterior cell open
  - (a) Eyes hairy—*Dialomusneura* Rondani (subgenus *Dialomusneura*)
  - (b) Eyes bare—*Dialomusneura* Rondani (subgenus *Coriopsis* Rondani)

colour, and are marked with purplish spots and streaks which

W. L. WEDDERSLEY, F. R. S. E. & F. I. C. & F. I. C. & F. I. C.

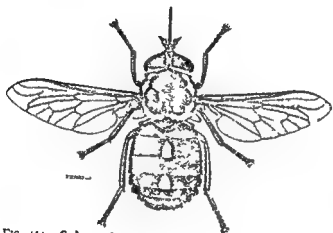


FIG. 414.—*Cadicera chryso stigma* WIEDEMANN FEMALE ( $\times 4$ )

#### *Silvius* V. 1820

This widely distributed genus may be exemplified by *Silvius fallax* Austen, 1912, found in North Eastern Rhodesia and *S. decipiens* Loew. The characters of the antennae are illustrated in Fig. 409 a, a', a'' a'''

#### *Cadicera* Macquart, 1854

Over a dozen species of this genus are now known in the Indian region, of which the following may be mentioned *C. melan*



## BRACHYCERA HETERODACTYLA

- A Antenna apparently two jointed with a three jointed arista  
Small hunch backed quick running flies—*Phoridae*
- B Antenna always three jointed  
Empodia wanting vestigial or linear
- I Radial 4 and 5 separate
- (a) Arista dorsal—*Empidæ* (in part)
- (b) Arista terminal
- (1) Front hollowed out between the eyes Males never holoptic Proboscis without fleshy labelle at tip—*Asilidæ*
- (2) Front plane or convex Males often holoptic Not more than four posterior cells Third antennal joint without bristle or style—*Scenopinidæ*
- II Radial 4 and 5 not separate  
Wings not lanceolate anal cell short second basal cell confluent with distal cell Not brilliantly coloured—*Empidæ* (in part)

The orders *Therevidæ* *Midasidæ* and *Dolichopodidæ* while containing species predatory on other insects do not appear to attack man

## FAMILY ASILIDÆ

## FAMILY PHORIDÆ

*Phora fessorata* occurs occasionally in houses *Aphiocheta ferruginea* Bruner causes intestinal myiasis

## FAMILY SCENOPINIDÆ

*Scenopinus fenestralis* Linnæus is the so-called window fly which is probably the only household fly which is not injurious to health

## FAMILY EMPIDÆ

It is doubtful whether these insects attack man As a rule they live on the  
as of other insects and plants

is also unknown

in Northern Rhodesia and *D* (*Corizoneura*) *hasla* Austen 1911  
in Portuguese East Africa

#### Rhinomyza Wiedemann 1820

In this genus the first and second segments of the antennae are short while the third segment is composed of five rings (Fig 108).  
Moreover the proboscis is short. The pro-  
boscis is 5-6 times the length of the antenna. The pro-  
and *R. detrita* known in Java  
Iocw *R. costalis*  
be noted as may *R. umbraticola* Austen 1911 in North Eastern  
Rhodesia and Kalanga while *R. maculata* Surcouf is found in  
Madagascar

#### Other Genera

Other genera of the Pangoninae are *Dicranis* Macquart in Brazil  
*campta* Schiner in Australia *Ptilocera* Tos in Central America *Contop*  
Africa in North Am  
America *Apalofestes*  
Africa *Sciens* Walke  
*Gastroides* Saunders

#### FAMILY LEPTIDÆ

*Orthorrhapha brachycera* with brownish medium sized or long narrow  
bodies and small heads. The third antennal joint is short and carries a  
a terminal brush or bristle. The proboscis resembles that of the Tiban  
it does the wing venation

Three genera are accused of blood sucking *Leptis* *Symphoromyza* and  
*Trichopterus*

and *L. strigo*  
*Symphoro*  
and *tibia* and  
*Trichopte*

but the oth

*philus* *Æstrus* *Hypoderma* etc and (b) *Cuterebrinae* with a well developed retractile proboscis including *Dermatobia* etc The four genera of interest to us may be recognized as follows —

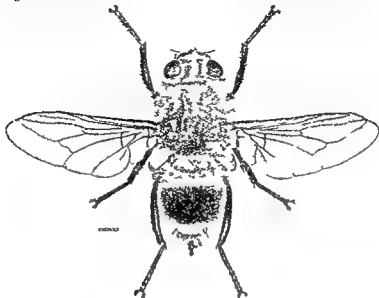


FIG 416—*Hypoderma bovis* DE GEER ( $\times 2\frac{1}{2}$ )

I Wing without posterior transverse vein the media runs towards the posterior border First posterior cell is partly open

Arista naked—*Gastrophilus* Leach 1817

II Wing with posterior transverse vein The media at its end is bent towards the radius and the first posterior cell is either open partially open or closed

A

Tris-  
ody

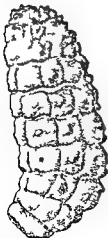


FIG 417—LARVA OF  
*Hypoderma bovis*  
DE GEER ( $\times 2\frac{1}{2}$ )

B Facial grooves remote

(a) Proboscis straight capable of being extended or entirely rudimentary Antennal groove with small angular dividing wall Palpi absent—*Hypoderma* Latreille 1825

(b) Proboscis bent and capable of being drawn into a deep cavity on the under surface of the head and generally hidden there Arista with hairs on the upper side Third antennal joint much longer than the first two—*Dermatobia* Brauer 1860

## SUBORDER II CYCLORRHAPHA

Section 1 *Aschiza*—This group includes the family Syrphidae of which no species is known to bite man

Section 2 *Schizophora*—This group includes the true flies characterized by a distinct frontal lunule and a frontal suture antennæ with three simple segments and an arista which is generally dorsal. They may be classified into—

## Muscoidea

Synonym—*Eumyidae*

This superfamily is divided into—

TRIBE 1 *Muscoidea acalyptratae* without squamæ covering the halteres (see Chapter XXV)

TRIBE 2 *Muscoidea calyptratae* with squamæ covering the halteres.

## MUSCOIDEA CALYPTRATAE

- 1 *Cestridae*
- 2 *Sarcophagidae*
- 3 *Muscidae*
- 4 *Anthomyidae*

## DIAGNOSTIC TABLE

- A First posterior cell of the wings not widely open
- I Antennæ small more or less hidden in round pits arista single or plumose body very hairy—*Cestridae*
  - II Antennæ well marked not hidden arista more or less plumose body not very hairy
    - (a) Arista plumose for only half its length bare in the terminal half which is hair like—*Sarcophagidae*
    - (b) Arista plumose or pectinate along its whole length—*Muscidae*
- B First posterior cell widely open—*Anthomyidae*

## FAMILY CESTRIDE

*Muscoidea calyptratae* with very hairy bodies which cause them

time after the other on all these forms and it is

Strahan Kolb Nagal Arnold and Smith and others but whether these are *D. cyaniventris* or some other species has not been determined

**Pathogenicity**—They cause pain and itching at the infected spot with swelling and œdema of the surrounding region giving rise to a boil like swelling rather hard of a deep red colour with a central opening Berne or ura of Brazil is the disease

**Treatment**—On inspecting the region a small opening will be seen and the larva may be noted showing its stigmata at times and at other times disappearing and reappearing like a jack in the box There is no difficulty in seizing this parasite with a pair of forceps and forcibly removing it and then treating the wound antiseptically The Brazilians try to asphyxiate the larva by tobacco smoke or apply some animal fat to the opening in the little tumour The fat is said to act by preventing the larva from breathing and compelling it to leave the tumour Some authors advise the application of calomel to the opening

#### *Dermatobia* (?) *kenzæ* Kolb

Kolb described a reddish brown fly in East Africa under this term

The fly behaved like *D. cyaniventris* in laying its eggs on the skin of people when bathing The larvæ entered the skin forming nodules The natives called the fly ngumba It is however possible that this is not a true *Dermatobia* which is a New World genus but a *Cordylobia*

#### FAMILY SARCOPHAGIDÆ

*Muscoidea calyptrata* with large bodies and antennal bristles feathery at the base but hair like and very fine at the tip Legs stout First posterior cell closed or only slightly open

The Sarcophagidæ are the blow flies of which *S. carnaria* is common

Genera—*Sarcophaga* *Wohlfahrtia* *Sarcophila* *Cynomysia*

#### *Sarcophaga* Meigen 1826

It is by no means uncommon in the tropics to find ulcers and

*Hypoderma*—A good example of dermal infection is *Hypoderma bovis* de Geer, which infests cattle. The eggs are laid on the skin of the animal, and are probably transferred to the mouth

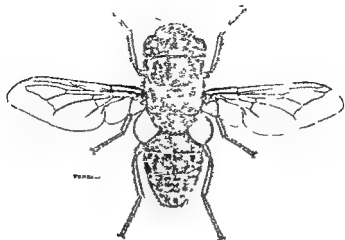


FIG. 418—*Tsetse* *ovis* LINNÆUS, FEMALE (X4)

by licking, whence they pass via the œsophagus to the skin in which they form tumours. It is found in Europe and America, and Peiper has gathered together histories of several cases in which *Hypoderma bovis* and *H. diana* Brauer have occurred in man but this is very rare. The larva which is commonly found in man belongs to *Dermatobia cyrenensis* which is described below.

*Osirus*—Rhinal myiasis is exemplified by *Osirus ovis* Linnaeus 1761, which has been found several times in the nasal cavities of



FIG. 419—LARVA OF *Osirus ovis* LINNÆUS (X4)

and transferred to the mouth and so to the stomach by licking. In this organ they live on the secretions and contents but though they do not bite the mucosa, they cause irritation. In due course they pass out of the alimentary canal with the feces, and then



*violitris* Lallen 1810, *S. haematodes* and *S. chrysostoma* Wiedemann 1839 *S. lambens* and *S. ruficornis* cause cutaneous myriasis in South America. *S. phillipsia* and *S. lambens* have been found in ulcers.

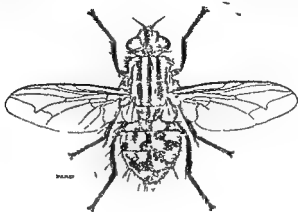


FIG 45—*Sarcophaga carnaria* LINNEUS FEMALE (X3)

Wohlfahrtia Brauer and Bergenstamm 1849

The larvae of *Wohlfahrtia magnifica* Schiner 1862, have been found in cavities in man's body and also in cattle, sheep, dogs, and domestic fowls.

*Sarcophila* Rondani 1856

The larvae of species of this genus—e.g. *S. meigeni* Schiner *S. latifrons* Fall and *S. ruralis* Fall—may also be found in ulcers etc in Germany, Austria Hungary, and France.

*Cynomyia* Robineau Desvoidy 1833

*Cynomyia mortuorum* Linnaeus 1761 is believed to infect ulcers with its larva.

#### FAMILY MUSCIDÆ

*Muscoides calyptata* with stout bodies and short thoraxes. Arista either entirely plumose or pectinated. The first posterior cell is either only slightly opened, or else closed at the border of the wing.

The Muscidæ include a number of important genera which may be recognized by the following table—



FIG 46—  
LARVA OF  
SARCOPIAGA SP.



- A Proboscis long adapted for biting—*Philæatomyia*
- I Proboscis partly chitinous partly fleshy, with large fleshy labellæ—*Philæatomyia*
- II Proboscis entirely chitinous with attenuated labellæ—*Stomoxys*
- (A) Arista feathered dorsally only
- (a) Palpi thin much shorter than the proboscis Third longitudinal vein with bristles proximally first posterior cell open—*Stomoxys*
- (b) Palpi thickened as long as or nearly as long as the proboscis
- (1) Proboscis long and tapering first posterior cell widely open third longitudinal vein without bristles—*Lyperosia*
- (2) Proboscis short and stumpy first posterior cell narrowly open third longitudinal vein
- (c) length swollen at the base Arista with many branched hairs Media (fourth longitudinal) with two sharp bends—*Glossina*
- (B) Arista feathered dorsally and ventrally
- (a) First posterior cell narrowly open third longitudinal vein without bristles *Hæmatobosca*
- (b) First posterior cell widely open
- (1) Third longitudinal vein with bristles proximally—*Hæmatobia*
- (2) Third longitudinal vein without bristles proximally—*Bdellolarynx*
- B Proboscis short not adapted for biting—*Muscina*
- (a) *usca*
- (b) not lustrous—*Calliphora*
- (2) Thorax and abdomen green or greenish lustrous
- (a) Scutum longitudinally marked—*Chrysomya*
- (b) Scutum not so marked
- (A) Thorax metallic green or bluish green—*Li cilia*
- (B) Thorax brassy green or purplish blue—*Pycnosoria*
- (3) "

## PHYLÆMATOMYINÆ

*Phylæmatomyia* Austen, 1909

Stomoxydinæ resembling *Musca domestica* Linnæus, being grey flies with remarkable proboscis. Front in male narrow, its width in centre being from one-eleventh to one-fifteenth of total width of head, width of the front in the female one-third of total width of head. Proximal portion of proboscis shows a swollen chitinous bulb, distal portion soft and fleshy and folded back under distal portion.

**Type Species.**—*Phylæmatomyia insignis* Austen, 1909

*Phylæmatomyia insignis* Austen, 1909

Body grey, with dorsum of thorax black. Larvæ are laid in batches of 2 to 22. Egg is 2 to 2.2 millimetres long. Larvæ hatch in eight to nine hours and when mature measure about 1.25 centimetres. They are bright lemon-yellow.

is ready to fly only occasionally from the

**Other Species.**—*Ph. lineata* Brunetti 1910 (synonym, *Pristirrhynchomyia lineata* Brunetti 1910) and *Ph. gurneyi* Patton and Cragg, 1912 both in India.

## STOMOXYDINÆ.

*Stomoxys* Geoffroy, 1764

Antæ of three segments, feathered dorsally only, proboscis long, tapering, chitinized in all its extent, non-retractile, palpi slender, very short, less than half the length of the proboscis. Fourth longitudinal vein curved so as to merely narrow the first posterior cell distally, third longitudinal vein bristly at its proximal end. Front narrower than the

S  
distribution The

**European Species.**—*S. calcitrans* L., 1758

**Asiatic Species.**—Twelve species known and two synonyms. *S. brunneipes* Grunberg, *S. calcitrans* L., *S. plurinotata* Bigot, *S. discus* Speiser, *S. indica*

Picard *S limbata* Austen *S nigra* Macquart *S bengalensis* Picard *S oblongo*  
*punctata* Brunetti *S pratensis* Summers *S pulla* Austen *S silens* Rondani

Speiser

Jo

The following table modified from Grunberg gives the diagnosis of a few of the species —

- A Legs entirely yellow Wings yellowish—*S laniatus* Bigot 1887  
 B Legs either entirely black or largely black or blackish brown Wings

I

73

II

omega mark on the

III

r part black without any markings—*S inornata* Grunberg 1906

- 2 Thorax with clear black stripes Abdomen with regular dark

(a) knee

(1)

two small brown longitudinal median lines — *S bilineata* Grunberg 1906

(2) Tibia and tarsus brown on all three legs Thorax with broad black longitudinal stripes

Wings blackish head and thorax brown yellow—*S brunnipes* Grunberg 1906

Wings glassy head and thorax grey—*S stellata* Grunberg 1906

(b) Legs black with light brown knees or decidedly dark brown spots

(1) Wing brushes yellowish brown with black tips Abdomen with dark middle longitudinal line Second

(2) W

*S calcitrans* Linnæus 1761

*Stomoxys calcitrans* Linnæus 1758

*S calcitrans* is the common stable fly found in houses stables and in the open near cattle It bites all classes of mammals and

## PHILÆMATOMYINÆ

*Philæmatomyia* Austen 1909

Stomoxydinæ resembling *Musca domestica* Linnæus being grey flies with remarkable proboscis. Front in male narrow its width

ular  
tion

Type Species.—*Philæmatomyia insignis* Austen 1909

*Philæmatomyia insignis* Austen 1909

ur with dorsum of thorax

eggs are laid in batches of

Egg is 2 to 2.2 millimetres

Larvæ hatch in eight to

nine hours and when mature measure about 1.25 centimetres

urrow under the

The puparium

is 0.18 centimetre

blood it is of a light mahogany colour and has eleven segments

The life-history may be summarized Egg laying five to ten minutes

egg eight to ten hours larvæ two days pupa three and a half to

to 2 2 2 2 2

1912 both in India

## STOMOXYDINÆ

*Stomoxys* Geoffroy 1764

Antæ of three segments feathered dorsally only proboscis long tapering chitinized in all its extent non retractile palpi slender very short less than half the length of the proboscis Fourth

European Species—*S. calcitrans* L. 1758

Asiatic Species—twelve species known and two synonyms *S. brunipes*

Grünberg *S. calcitrans* L. *S. pluviosata* Bigot *S. dacnusa* Spe ver *S. indica*

coloured flies with the wings closed flat over one another and

*Nemorkina palpata* Their bites have been long known to be dangerous to animals but it was not till Bruce showed that they

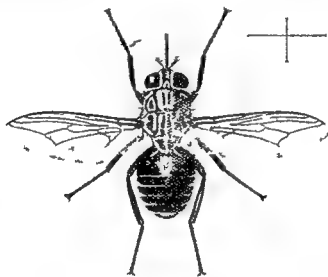


FIG. 429.—*Glossina palpata* FEMALE

mechanical

cause of the  
much attention  
in 1903 Bruce  
*T. castellani*  
attention was not

much attention

The eyes are large and bare and between them the vertex is depressed and at its back carries ocelli. In front a deep facial pit

specially  
to be the

cognized  
rejecting  
at their

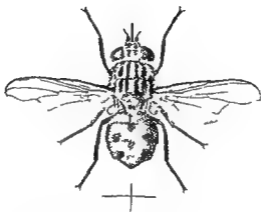


FIG 427 — *Stomoxys calcitrans* FEMALE

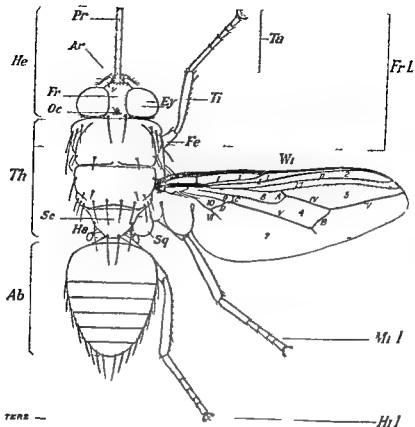


FIG 432—DIAGRAM OF A TSETSE FLY

(Modified after Austen from Monograph of Tsetse Flies )

*He* head *Th* thorax *Ab* abdomen *Fr l* front leg *M1 l* middle leg  
*H1 l* hind leg *Pr* proboscis ensheathed by the palpi *Ar* arista  
*Fr* frons *Oc* occiput *Ey* eye *Sc* scutellum *Ha* haltere *S1* squama  
*W1* wing *Fe* femur *Ti* tibia *Ta* tarsus

*Venation of Wing*—*Ia* Subcostal or auxiliary vein *1a 1b* two portions of the costal cell divided by the humeral transverse vein *1c* subcostal cell

I  
l  
E  
C  
V  
V  
I

cell *V1* anal or sixth longitudinal

and outwards to join with the posterior transverse vein when it again turns and runs obliquely forwards to join the costal vein just in front of the tip of the wing Between it and the radius 4 and 5 (third

is separated by a transverse impression from the gena The pro-

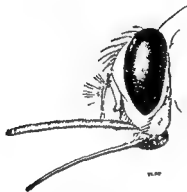


FIG 430—HEAD OF A GLOSSINA

This figure shows the proboscis being lowered previously to piercing the skin to suck blood

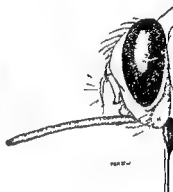


FIG 431—HEAD OF A GLOSSINA

This figure shows the proboscis ready for sucking

well marked and the subcostal vein (auxiliary) joins it about the junction of the inner with the outer third in the extended position of the wing thus enclosing the costal cell which is divided into two portions by the humeral transverse vein. The radius 1 (first longitudinal) curves forwards joining the costa about the junction of the inner two-thirds with the outer one third thus forming a very narrow subcostal cell. The radius 2 and 3 (second longitudinal)



The labium or second maxilla starts from the ventral area of the head and is first swollen to form the bulb. Anteriorly it is grooved dorsally to hold the hypopharynx and the labrum while farther anteriorly it ends in the labellæ. These structures (labellæ)

are joined together in the ventral line except anteriorly where there is a V shaped notch while dorsally they possess teeth which interlock.

In the ventral line the chitinous floor of the labium is prolonged forwards in the form of a fork in front of which is a membranous area anteriorly to which the inner wall of each labellum becomes divided into dorsal median and ventral segments. Each segment is armed with a series of about ten rasps composed of some thirty minute bars in front of which are two pairs of teeth. Between the bases of the anterior pairs of teeth there projects a fan shaped mass of spine like scales.

The segments are capable of eversion when the muscles pull backwards the external walls of the labellæ. In this way the teeth would be brought in contact with the skin and the wound necessary for blood sucking made probably by rotatory movements.

**Internal Anatomy**—The internal anatomy has been carefully studied by Minchin and does not materially differ from that of *Stomoxys*.

The pharyngeal tube opens into the pharynx which is situated mainly in the rostrum. Its walls are strongly chitinized forming the fulcrum. The œsophagus runs upwards and then backwards to open into the proventriculus and to be continuous via a long ventral duct with the crop which lies in the first two segments of the abdomen. The chylific ventricle is narrow at first in the thorax but becomes wider in the abdomen

where it coils several times. There are the usual ileum colon rectum and Malpighian tubules.

The salivary glands are two long coiled tubes lying first in the abdomen and then passing into the thorax and probably opening finally on the hypopharynx though this has not yet been worked out.

**Life-History**—The species of *Glossina* live in jungles or bush

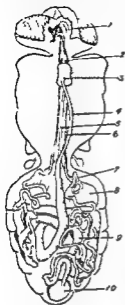


FIG 134 — ANATOMY OF A TSETSE FLY

(After Minchin from Reports of the Royal Society)

- 1 Pharynx 2 œsophagus 3 proventriculus 4 thoracic intestine 5 duct of sucking stomach 6 salivary duct 7 salivary gland 8 sucking stomach 9 abdominal intestine 10 rectum

longitudinal) lie internally the first (anterior) basal cell and externally the first posterior cell separated by the anterior transverse vein which is very oblique. The cubitus 1 and 2 (fifth longitudinal) joins the anterior basal vein marking out the posterior basal cell and then runs forwards to join the posterior transverse vein marking out the discoidal cell which is hatchet shaped with the handle running up to the anterior basal transverse vein. After this the vein turns backwards to join the margin of the wing

tapering to the apex and clothed with short black hairs. The male genitalia are characteristic. The ventral plate of the sixth segment carries a patch of dark hairs on each side of the middle line behind which is the hypopygium which is oval tumid and marked by a vulviform median groove the anus running from its anterior margin backwards to beyond the middle.

The proboscis shows the usual rostrum or conical head projection the haustellum or proboscis proper and the labelle.

The proboscis proper is com

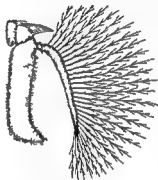


FIG 433 — ANTENNA OF A GLOSSINA SHOWING THE ARISTA

(After A sten from Tsetse Flies)

Farther forwards it separates from the labrum and ends in a point. The tube composed by the hypopharynx and the labrum is the afferent blood or pharyngeal tube. The hypopharynx starts below the pharynx surrounding the salivary efferent duct and pierces the bulb of the proboscis appearing on its dorsal aspect. Farther forward it lies in a groove on the labium articulating laterally with the labrum. Finally it ends as a delicate chitinous tube just posterior to the labelle. Its canal is the salivary tube or hypopharyngeal canal.



potami According to Kinghorn, copulation in *G morsitans* lasts for several days and may be completed in the abdomen and it cor



FIG 435—A GLOSSINA IN THE ACT OF GIVING BIRTH TO A LARVA  
(Modified after Newstead)

coloured, are to be found as a rule near the roots of banana trees. These larvæ are composed of twelve segments, the anterior of which carries the two minute mouth hooks, and the posterior a dark hood or anal segment. The larvæ retire to some hole, and in a few hours become jet black pupæ, from which the fully developed insects issue in about six weeks.

through which the imago escapes. The first segment carries the mouth and the twelfth two lateral tumid tuberculated lips connected by dorsal and ventral ridges enclosing a pit in which the posterior stigmata can be seen.

The larvæ of *G palpalis* measure 4.5 by 1.75 millimetres, and the pupæ 5 to 5.75 by 3 millimetres.

According to Kinghorn, forty-seven to fifty-three days elapse from the birth of the larva to the escape of the imago in *G morsitans*.

**Bionomics.**—As *G palpalis* is most probably the sole means of transmission of the



FIG 436—PUPA OF A TSETSE FLY

(After Austen from Tsetse Flies)

a Magnified  
natural size

some idea of what is carefully studied by

in bush near water, especially in the undergrowth composed of shrubs, bushes, vines,

head, body, and wing The thorax is marked dorsally by

in the black colour of the third and fourth abdominal (except margin) segments, and in the blackish quadrangular median on the second abdominal segment. In the male the eyes join

*Cordylobia rodhaini* Gedoelst, 1905.

Synonym.—Lund's larva.

Under this term a larva is described as occurring in the Free State which possesses habits similar to those of *Cordylobia anthropophaga*

The larva of this species also occurs at times under the skin of man in the Belgian Congo

*Auchmeromyia* Schiner and Brauer Bergenstamm, 1819

*Auchmeromyia luteola* Fabricius, 1805.

Synonym.—*Musca luteola* Fabricius 1805 Ver de Case.

The larva of this fly is called the Congo floor maggot. It was first found by Dutton Todd, and Christy living in the floor of huts to the depth of 3 inches. At night these larvæ came out and sucked the blood of persons sleeping on the ground, or on beds little raised therefrom, but not on high beds

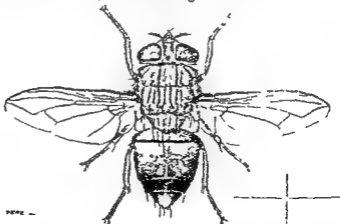


FIG 441.—*Auchmeromyia luteola* FABRICIUS FEMALE (X3)

The native names for the larvæ are, Mabinzu, Nch Ntunga, Mvudi and kiso

Morphology.—The fly is widely distributed in tropical and tropical Africa. It is 10 to 12 millimetres in length, tawny colour, with small black hairs giving it a smoky appearance. The head is as broad as the thorax. The eyes are separated

*Cordylobia* Grünberg, 1903*Cordylobia anthropophaga* E. Blanchard

Synonyms.—*Ochromyia anthropophaga* E. Blanchard, *Glossina grunbergi* Donitz

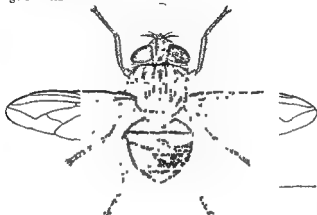


FIG 439—*Cordylobia anthropophaga* BLANCHARD FEMALE (X4)

The larva of this fly, which is called ver du Cayor because it was first noticed in Cayor, in Senegambia burrows into the skin and causes a painful swelling. It extends from Senegal to Natal.

**Life-History and Morphology.**—It is not certain whether the fly

the clothing and simply enters the skin.

The larva, which measures about 12 millimetres, is composed of twelve segments of which the anterior or cephalic is bluntly pointed in front and truncated behind, and carries two black mouth hooklets on its ventral surface. External to these hooklets lie the antennal protuberances. On the third to the eleventh segments there are minute, rather characteristic, brownish chitinous, recurved spines. The greatest breadth is at the level of the sixth to seventh segments, while the last segment has a flattened posterior surface which carries the posterior stigmata. It is parasitic in men, monkeys, and dogs. The pupa measures 10.3 by 4.6 millimetres and looks like an ordinary muscid pupa.

The fly measures 9.5 millimetres, and has a yellowish coloured



FIG 440—LARVA OF *Cordylobia anthropophaga* (X4)  
(After Austen)

**Auchmeromyia prægrandis** Austen 1910

A saffron yellow fly. It occurs in South Africa

**Pollenia** Robineau Desvoidy, 1830

The larvæ of *Pollenia rudis* Robineau Desvoidy have been found in a case of gastric myiasis in man

**Bengalia depressa** Walker

By an error this fly was said to cause cutaneous myiasis in Natal Rhodesia British Central Africa Uganda and the Sudan the true causal agent being *Cordylobia anthropophaga* Grunberg. The life history of *B. depressa* is unknown

## FAMILY ANTHONYIDÆ Latreille

Diptera with arista naked or pectinate. Thorax with complete transverse suture. First posterior cell completely open. Abdominal bristles often absent.

**Fannia** Robineau Desvoidy 1830**Fannia canicularis** Linnæus 1761

Synonyms -- *Homalomyia canicularis* Linnæus *Anthomyia canicularis* Linnæus

This species has frequently been reported as being passed in human feces

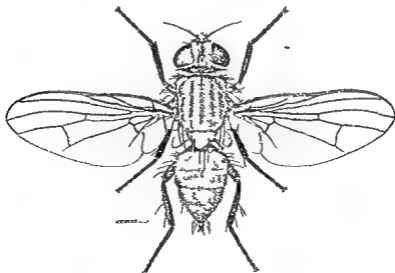


FIG 443—*Fannia canicularis* LINNÆUS FEMALE (X8)

brown stripes and shows a well marked transverse suture. The squame are large yellow in colour and cover the halteres. The first abdominal segment has a narrow dark line posteriorly, the second a central median dark line which joins with a posterior dark line. The third segment is dark brown except for a narrow yellow anterior streak. The fourth segment is dark coloured with a posterior light brown band. The fifth segment is small and con-



FIG. 442.—*Auchneromyia luteola* LARVA  
( $\times 3$ )

ventral surface is flattened and has three footpads transversely arranged at the posterior margin of each segment. The last segment is large and carries the posterior spiracles and the anus.

The mouth which is provided with teeth leads into an oesoph-

three weeks.

**Habits**—The fly does not bite man. The larva as described above attacks man and fills its dorsal oesophageal pouch with blood and thus acquires a red colour.

**Pathogenicity**—As far as is known it is non pathogenic.

In 1911 Roubaud described a new genus *Charomyia* Roubaud 1911 of which the larvae of two species *C. bo sets* Roubaud 1911 and *C. charophaga* Roubaud 1911 were blood suckers attacking African wart hogs and African ant eaters.



with regard to an African myiasis called muculo but as far as this disease has not been traced. Wellman says that the term is

*Hydrotaea* Robineau Desvoidy 1830

*Hydrotaea niteorica* Linnæus which usually attacks animals nostrils is said to attack man also

**SUBORDER III PUPIPARA**

Synonym — *Proboscidea*

The Pupipara are flies which

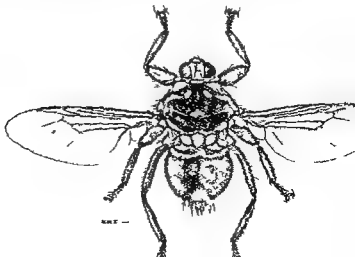


species either entirely or after

They do not lay eggs but produ

FIG 446 — NYCTERIBIA SP (?)

The Nycteridae are parasitic on birds and bats. In this family *Penicillidia dufouri* Westwood which is the carrier of *Achromaticus* is Dionisi 1898. The Braconidae are parasitic on bees and the on bats and therefore will not be considered further



## FANNIA GANICULARIS

but instead of diarrhoea constipation resulted with severe headaches. The abdomen was distended.

The patient was treated with raw pumpkin seeds and then given a saline purge and passed 1,000 to 1,500

authentic cases of myiasis of the urinary passages caused by the larva of this fly.

### *Fannia desjardensis* Macquart

**Synonyms**—*Homalomyia desjardensis* Macquart & *Homomyia desjardensis* Macquart

Wellman describes cases of this myiasis in the urinary canal of human beings in Angola who

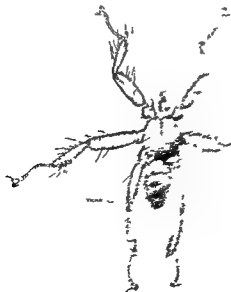


FIG. 445.—*Fannia ganicularis* larva.

with regard to an African myiasis called *maculo* but as far as we know the term is not used in seven to nine days

*isurata* / *manicula*

*F. saltatrix* as being causes of intestinal myiasis.

### *Hydrotaea* Robineau Desvoidy 1830

*Hydrotaea meteorica* Linnæus which usually attacks animals eyes and nostrils is said to attack man also

### SUBORDER III PUPIPARA

#### Synonym — *Eproboscidae*

The Pupipara are flies which appear to have become altered owing to their parasitic life. They possess a well defined proboscis which is said by Austen to resemble that of the *Glossinæ* by being armed at its tip with teeth. Wings have been lost in several species either entirely or after the imago has become parasitic though they may exist throughout life in other species. Their feet are provided with extra unguis to enable them to cling to the hairs etc of the host. They do not lay eggs but produce a larva which soon becomes a pupa.

The suborder is divided into four families (1) *Hippoboscidae* (2) *Nycteribidæ* (3) *Braulidæ* (4) *Streblidæ*

The *Nycteribidæ* are parasitic on birds and bats. In this family comes *Pencilidia dufouri* Westwood which is the carrier of *Achromaticus vesperugo* Dionis 1898. The *Braulidæ* are parasitic on bees and the *Streblidæ* on bats and therefore will not be considered further.



FIG 446 — *NYCTERIBIA* SP (?)

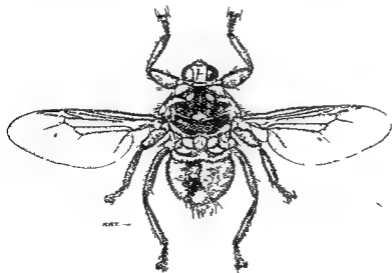


FIG 447 — *Hippobosca rufipes* VON OLFERS FEMALE (X4)



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to *Piophilis casesi*)

HIPPOBOSCIDÆ

FAMILY HIPPOBOSCIDÆ

In one jointed  
the tarsus is



FIG 448 LARVA AND PUPA OF *Hippobosca rufipes* VON OLPERG (X4)

FIG 449—HEAD OF *Hippobosca rufipes* FEMALE

*Hippobosca equina* Linnæus is known on horses in the New Forest England  
*Hippobosca capensis* Leach on dogs in Africa India Persia and South Europe  
*H. camelina* Leach and *H. maculata* Leach are found in the Sudan and Egypt  
and in addition to these *Melophagus ovinus* Linnæus is found all over the world

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res vii in 284  
Bdellolearynx

Muscid larvæ )

□  
Insect . . 7 vols

Hamm

Præpar. Insect. Pl. 100

ring<sup>4</sup>  
longe  
the l

continue forwards on to the frons and probably delineate the four segments of which the head is composed

Anterior and ventral to the frons and gena lies the perioral ring which carries the mouth appendages which consist of a labrum (epipharynx) mandibles maxillæ and palps hypopharynx and labium with palps. There does not appear to be a separate clypeus.

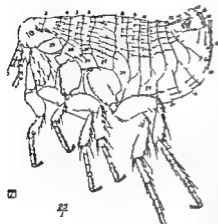


FIG 450—*Xenopsylla cheopis* MALE  
(After Jordan and Rothschild *Journal of Parasitology*)

1 Labrum mandibles and labial palps

notum 7 12 ordinary abdominal tergites 13 seventh tergite with bristle (behind this is seen the small eighth

sternite

bulo basal articulation and an anterior portion which projects from the

The labrum (variously known as epipharynx hypopharynx and by other names) is a hollow prolongation of the dorsal wall of head and pharynx. In front it is closed while behind it opens into the coelom. Ventrally it shows a groove converted into a canal when it is articulated with the mandibles laterally. The mandibles consist of basal segments attached to the sides of the mouth and an anterior portion which projects freely forwards and shows fine serrations anteriorly. The inner aspect of the mandibles possesses a groove converted into a trilobed channel by articulation with the fellow of the opposite side and the hypopharynx.

The maxillæ are triangular chitinous plates each possessing a four-jointed maxillary palp. The labium (which of course represents the second maxillæ with their palps) is

other mouth parts

The hypopharynx consists of a basal portion

## CHAPTER XXXIV

# SIPHONAPTERA AND COLEOPTERA

Siphonaptera—Sarcopsyllidæ—*Dermatophilus penetrans*—Pulicidæ—Pulicinæ  
—*Pulex irritans*—*Xenopsylla cheopis*—Coleoptera—Orthoptera—  
References

### SIPHONAPTERA Latreille 1825

Synonyms.—*Rophotaera* Schellenberg, 1798, *Aptera* Lamarck, 1801;

lateral plate like appendages on the meso- and meta-thorax.

The antennæ are three-jointed, and embedded in grooves. The third joint has nine more or less separated pseudo-joints.

Remarks.—Fleas have come into considerable prominence, owing to the work of the Indian Plague Commission and that of Dr Verbitski, of St Petersburg, who have shown that they are to be looked upon as the main agents by which plague is spread from rat to rat, and from rat to man.

Fleas may also carry blood-parasites—as, for example, *Trypanosoma leucis* and — — — — —  
thi

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ma

berg in 1880, and the third by Baker in 1904.

Recently much work has been done on these parasites by Rothschild and Jordan.

Morphology.—The head is small and may or may not possess eyes which, when present are *entirely* *absent* *behind* *the* *head* *is*





and the epimerite. The episternite has the anterior and ventral port on separated off by an oblique incrustation to form a sternite. The prosternite or sternite of the prothorax is not divided. Generally these various sclerites show bristles.

different segments must be considered according to sex. In the female the eighth tergite is very broad ventrally but the sternite is reduced to an elongated plate lying between the ventral edges of the tergite.

The ninth tergite carries the sensory plate with usually fourteen (there may be more) setiferous grooves. The ninth sternite is membranaceous laterally and extends far ventrally where it is strongly chitinized and lea

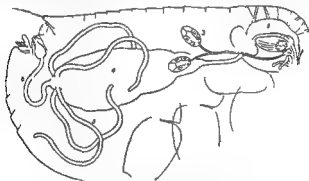


FIG 451 — ANATOMY OF A FLEA

(From the *Journal of Hygiene* 1906)

- 1 Mouth 2 pharynx 3 salivary glands 4 stomach 5 Malpighian tubes 6 intestine 7 rectum

in the h h - I

at the apex

swollen into a bulb which represents the proventriculus

At the junction of the stomach with the intestine are the openings of the

segments posteriorly The head is in the bottom of the burrow

ten days

Pathogenicity.—This will be described later (Chapter XCVI) but it may be mentioned that it includes irritation pus formation ulceration and formation of a sore, which may become infected with bacteria and cause loss of a toe or a leg or even tetanus may develop.

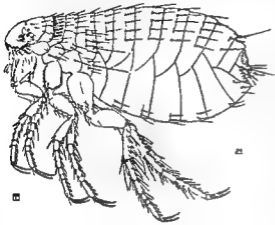


FIG 457—*Glenocephalus felis* BOUCHÉ

This drawing shows the combs on the head and prothorax

FAMILY PULICIDÆ  
Stephens, 1829

Siphonaptera with body compressed or elongated always larger than in the preceding family Head in comparison to the rest of the body small, top round, venter protected with hairs,

often no plate segment

men never so swollen that the original form is lost Female never endoparasitic

There are three subfamilies *Pulicinae* Tiraboschi *Tylopsyllinae* Tiraboschi *Hystrochopsyllinae* Tiraboschi

SUBFAMILY PULICINÆ

Pulicidæ with eyes

The more important genera of the Pulicinae may be arranged as follows (modified after Jordan and Rothschild) —

- A Ctenidia on prothorax and head—*Glenocephalus*
- B Ctenidia on prothorax and only two teeth on cheek at genal angle—*Christopsylla*
- C Ctenidia on prothorax only

## D Ctenidia absent on prothorax and head

- I Terminal segment of antennæ short only distinctly segmented posteriorly Hind coxa with a comb fifth tarsal segment

(1)  
(2)

tion of the coxa upward

(a) Anterior angle of genal edge prolonged backwards into a triangular lobe, pronotum stronger than metanotum  
—*Parodontis*

(b) Anterior angle of genal edge not produced into triangular lobe—*Xenopsylla*

(3) Mesosternite without intercal rod like incrasation from

II

(1) Terminal segment of antennæ symmetrical genal process with a number of bristles—*Parapsyllus*

(2) Terminal segment of antennæ asymmetrical proximal segments sloping backwards Genal process with only one to two bristles—*Phopalopsylla*

III Terminal segment of antennæ segmented all round symmetrical Hind coxa without comb fifth tarsal segment with five lateral bristles at least and subapical hair

(1) Ant ... ..

( )

## SUBFAMILY TYPHLOPSYLLINÆ

Pulicidæ with eyes absent or very rudimentary Head rounded in front Body thin

Genera.—*Ctenopsylla* Kolenati *Ctenophthalmus* Kolenati 1857, *Typhlopsylla* Wagner *Neopsylla* Wagner *Typhloceras* Wagner *Ctenophthalmus* can be recognized by having movable ctenidia in front of the ocella and the rest can be differentiated as follows —

I Th ... ..

II

III

IV

## SUBFAMILY HYSTRICHOPSYLLINÆ

Abdominal tergites with one or more ctenidia posterior tibial spines in numerous short, close set transverse rows on posterior border with about four spines in each row female with four antepygial bristles on each side

Genera.—*Hystrichopsylla* Taschenberg *Macropsylla*

## THE FLEAS OF RATS AND MICE

The following table gives the fleas observed on rats, mice, and field mice, by Taraboschi and Rothschild

segments posteriorly The head is in the bottom of the burrow

ten days

**Pathogenicity**—This will be described later (Chapter XCVI) but it may be mentioned that it includes irritation pus formation

ulceration and formation of a sore which may become infected with bacteria and cause loss of a toe or a leg or even tetanus may develop

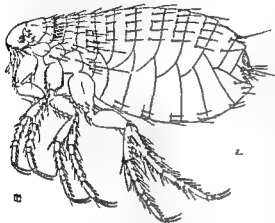


FIG 457—*Ctenocephal is felis* BOUCHÉ

This drawing shows the combs on the head and prothorax

#### FAMILY PULICIDÆ

Stephens 1829

Siphonaptera with body compressed or elongated always larger than in the preceding family Head in comparison to the rest of the body small top round venter protected with hairs

often no eyes The antennal groove is at times covered by a chitinous plate The end segment of the antenna is either segmented or unsegmented Thorax wide pronotum often with ctenidia Abdomen never so swollen that the original form is lost Female never endoparasitic

There are three subfamilies *Pulicinae* Tiraboschi *Tylopsyllinae* Tiraboschi *Hystriichopsyllinae* Tiraboschi

#### SUBFAMILY PULICINÆ

Pulicidæ with eyes

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A Ctenidia on prothorax and head—*Ctenocephalus*

B Ctenidia on prothorax and only two teeth on cheek at genal angle—

C

- D Ctenidia absent on prothorax and head
- I Terminal segment of antennæ short only distinctly segmented posteriorly Hind coxa with a comb fifth tarsal segment with four lateral and one subapical bristle
- (1) Forms with small mesial tubercle—*Macopsylla*
- (2) Forms without small mesial tubercle —
- (A) Mesosternite with internal rod like incrasation from insertion of the coxa upward
- (a) Anterior angle of genal edge prolonged backwards into a triangular lobe pronotum stronger than metanotum — *Paritodonta*
- (b) Anterior angle of genal edge not produced into triangular lobe—*Yenopsylla*
- (B) Mesosternite without internal rod like incrasation from insertion of the coxa upward—*Pulex*
- II Terminal segment of antennæ segmented all round hind coxa without a comb fifth tarsal segment with four lateral bristles and one subapical hair
- (1) Terminal segment of antennæ symmetrical genal process with a number of bristles—*Parapsyllus*
- (2) Terminal segment of antennæ asymmetrical proximal segments sloping backwards Genal process with only one to two bristles—*Phopalopsylla*
- III Terminal segment of antennæ segmented all round symmetrical Hind coxa without comb Fifth tarsal segment with five lateral bristles at least and subapical hair
- (1) \*
- (2)

## SUBFAMILY TYPHLOPSYLLINÆ

Pulicidæ with eyes absent or very rudimentary Head rounded in front Body thin

Genera—*Ctenopsylla* Kolenati *Ctenophthalmus* Kolenati 1857  
*Typhlopsylla* Wagner *Neopsylla* Wagner *Typhloceras* Wagner  
*Ctenophthalmus* can be recognized by having movable ctenidia in front of the ocelli and the rest can be differentiated as follows —

- I Third tarsal with five lateral bristles on each side—*Typhloceras*
- II Third tarsal with four lateral and two accessory bristles—*Ctenopsylla*
- III Third tarsal with four lateral and no accessory bristles—*Neopsylla*
- IV Third tarsal with three lateral and two accessory bristles—*Typhlopsylla*

## SUBFAMILY HYSTRICOPSYLLINÆ

Abdominal tergites with one or more ctenidia posterior tibial spines in numerous short close set transverse rows on posterior border with about four spines in each row female with four antepygial bristles on each side

Genera—*Hystrichopsylla* Gaschenberg *Macropsylla*

## THE FLEAS OF RATS AND MICE

The following table gives the fleas observed on rats mice and field mice by Tiraboschi and Rothschild

**Life-History**—The eggs are round waxy white or pearly in colour and resemble small seeds.

Larvæ which at first are very active become sluggish and ceasing to eat spin cocoons composed of fine white silk like fibres. The

two days

without a feed  
fed then it dies  
but *Xenopsylla*

*cheopis* will live forty one days on a rat and twenty seven days on a man. Therefore the whole life of a rat flea from birth to death is about sixty three days. A rat flea is more readily attracted by a rat than by a man. It breeds at all temperatures but has one optimum temperature above and below which it does not thrive so well. Dampness is injurious to the flea killing the larvæ and

the flea of  
*Xenopsylla*  
*cheopis*  
guinea

pigs cats rabbits antelopes kangaroos and men

The infection with plague bacilli does not appear to affect the flea's health for it has an immunity dependent apparently on phagocytosis.

### *Hoplopsyllus* Baker

Closely related to *Pulex* but distinguished at once by the ctenidia on the prothorax. *Hoplopsyllus anomalus* Baker is the plague carrier of the Californian ground squirrel.

### FAMILY CERATOPSYLLIDÆ Baker 1905

Siphonaptera with ctenidia present on the metathorax and abdomen. eyes rudimentary or absent.

Genus—*Ceratopsyllus* Kolenati

The species of this genus are found on bats.



FIG. 40.—LARVA OF A BEETLE PASSED PER URETHRAM  
(After King)

- (b) Anterior angle of genal edge not produced into triangular lobe—*Xenopsylla*  
 (2) Mesosternite without internal rod like incrasation from

II

( " " "

(

III

- (1) Antennal groove open behind  
 (a) Abdominal tergites with one row of bristles except first which bears two First hind tarsal segment shorter than second—*Coplopsylla*  
 (b) Abdominal sternites with very numerous short bristles First hind tarsal segment longer than second—*Goniopsyllus*  
 (2) Antennal groove closed behind  
 Abdominal tergites with one row of bristles—*Lycopsylla*

## SUBFAMILY TYPHLOPSYLLINÆ

Pulicidæ with eyes absent or very rudimentary Head rounded in front Body thin

Genera.—*Ctenophthalmus* *Typhlopsylla* *Ctenophthalmus*

*Typhlopsylla*

*Ctenophthalmus*

in front of the

I

II

III

IV

## SUBFAMILY HYSTRICHOPSYLLINÆ.

Abdominal tergites with one or more ctenidia, posterior tibial spines in numerous short, close set transverse rows on posterior border, with about four spines in each row, female with four antepygial bristles on each side

Genera.—*Hystriehopsylla* Taschenberg *Macropsylla*.

## THE FLEAS OF RATS AND MICE

The following table gives the fleas observed on rats, mice, and field mice, by Tiraboschi and Rothschild



## CHAPTER XXXV

# THE ANIMAL CARRIERS OF DISEASES

Preliminary—Historical—Protozoal diseases—Helminthiasis—Myiasis—Bacterial diseases—Diseases of unknown causation—Chance transmission—Imperfect carriage of parasites—Terms—References

### PRELIMINARY

THE present chapter is an attempt to put in concrete form the role of the animal carrier of disease.

Animals can produce traumatism<sup>s</sup> by their bites and can cause disease by injecting chemical substances manufactured in their bodies—e.g. American and Australian tick paralysis—but these questions do not now concern us. The problems which we are about to consider are those associated with the spread of diseases known or suspected to be parasitic. Such diseases are divisible into those caused by animal and those caused by vegetal parasites. The

we will consider first

A given animal parasite apparently has some form of *sexual generation* in some stage of its life history and it is probably merely our lack of knowledge which prevents us from acknowledging this as a proven fact.

The host in which the sexual generation takes place is called the *definitive host* and is probably the original host in which as a rule the parasite does not produce severe forms of disease and may produce no ill effects at all. This shows that it and its host have become so adjusted that it does not overproduce itself in the host which on its part does not poison or otherwise attack the parasite.

It is certainly not the object of the parasite to kill its definitive host, but to leave it by some route which causes no great disturbance of its tissues or functions. Hence intestinal parasites leave

definitive host, and the cycle may begin again but the dangers of the outer world may be guarded against by entering some animal's body in which no development occurs. Such an animal would be



*Rhodesiense* Type of Sleeping Sickness—Kinghorn and Yorke have described short blood trypanosomes in man and judging by Miss Robertson's Castellani experiments these must be the transmission agents which infect *Glossina morsitans* in the salivary glands of which short trypanosomes occur which infect the vertebrate which is the intermediate host

We now come to the very important question of the intermediate reservoir of this trypanosome Bruce says that *T. brucei* and *T. rhodesiense* are one and the same parasite Assuming this to be true the intermediate reservoir would be the African antelopes—e.g. *Catoblepas gnu* the wildebeest *Strepsiceros capensis* the loodoo *Taurotragus scriptus* var *sylvaticus* the bush buck But there are doubts about this because

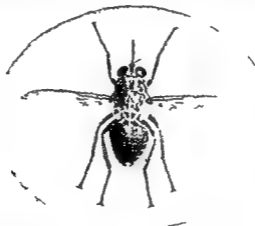


FIG. 462.—*Glossina morsitans* WESTWOOD 1850 THE CARRIER OF THE TRYPANOSOME OF THE STEPHENS AND FANTHAM TYPE OF SLEEPING SICKNESS

(From a photograph by J. J. Bell)

where the Rhodesiense form of sleeping sickness is unknown

2 Chalmers and O Farrell working with a posterior nucleated

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an  
nd  
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—

parasite this may grow invade its tissues and quite different from the intestines. The second host now becomes a true *intermediate host* but it is something relatively new interposed in the life-cycle of the parasite which has not yet adjusted itself to its new host nor has this host adjusted itself to the parasite and the result is that the parasite almost invariably causes disease in the intermediate host which may be a vertebrate or an invertebrate—e.g.—

## THE DEFINITIVE HOST

<i>Parasite</i>	<i>Definitive Host</i>	<i>Intermediate Host</i>	<i>Nature of Parasitism</i>
<i>Filaria bancrofti</i>	Man but little affected pathologically	Culex and Stegomyia mosquitoes severely affected by infection	True parasitism of the vertebrate
<i>Plasmodium malariae</i>	Anopheles mosquitoes unaffected pathologically	Man suffers from malarial fever	True parasitism of the mosquito

It is therefore obvious that these two diseases from the point of view of evolution have two quite different origins. The first is originally a parasite of man and as Hindle has pointed out Manson's original idea of water infection may be the true method and that *ab initio* the 'Larvofilaria' lived in water and pierced the human skin, as it does to day on leaving the mosquito and requires as Bahr has shown dampness in order to live while it pierces the skin. The mosquito carrier is therefore a relatively new acquisition and the mosquito not having adjusted itself to these conditions often dies as Bahr has shown. On the other hand *Filaria bancrofti* often dies as Bahr has shown. On the other hand *Filaria bancrofti* causes no symptoms in man but if there are accidents the disease ensues.

It is quite otherwise with the malarial parasites in which the

with this animal may also be a definitive host as may *Lamus infestans* and *L. sordida* while Brumpt has shown that *T. cruzi* can develop in *Chinocoris lectularius* and in *Leptocimex boneti* (It must be remembered that *Triatoma* can be infected naturally with a trypanosome)

There is no evidence of hereditary infection in these insects but there is some evidence that at times infection may be contaminative from the insect faeces via the bite but this requires more investigation

The chart of this disease would be —

### CHAGAS' DISEASE

<i>Parasite</i>	<i>Definitive Host</i>	<i>Infection</i>	<i>Intermediate Host</i>	<i>Intermediate Reservoir</i>	<i>Transmission</i>
<i>Trypanosoma cruzi</i>	<i>Lamus megistus</i> (synonym <i>Triatoma megista</i> )	Short sal vary try panosomes — Inoculative	Man	<i>Dasytus novemcinctus</i>	Male and female try panosomes — Ingestive

**Leishmaniasis**—The nature of the carrier and the reservoir is very uncertain at the present moment. Judging by the more marked resistance of the dog to experimental infection in India and the Sudan we may assume that there are at least two kinds of kala azar. It is believed by certain authorities that a flea is the transmitter of the Mediterranean type whereas the Indian and Sudan type are not so transmitted. Patton's incrimination of the bug has not stood the test of time. Archibald has suggested and brought forward evidence that at least in regard to the Sudan infection is probably due to the ingestion of cysts from water arthropods.

The development of generalized kala azar in Archibald's monkeys after the successful inoculation of Oriental sore points to a close relationship between the two diseases as suggested long ago by Manson.

There is no complete evidence at present that the espondia  
may  
ent

The present state of our knowledge which is unsatisfactory may be summarized as follows —

3 Laveran's cross immunity experiments mentioned in Chapter XIX show that *T. brucei* and *T. rhodesiense* are quite different from an immunity point of view

4 Taute has injected himself with ½ c c of blood from a dog infected with *T. brucei*. He did not become infected, and suffered no bad effects

5 Taute fed *Glossina morsitans* upon animals infected with *T. brucei* and after waiting the necessary time these flies were allowed to feed upon two men with negative results although

DECOUPE —

#### STEPHENS AND TANTHAM TYPE OF SLEEPING SICKNESS

Parasite	Definitive Host	Definitive Reservoir	Infection	Intermediate Host	Intermediate Reservoir	Transmission
<i>Trypanosoma rhodesiense</i>	<i>Glossina morsitans</i>	Hereditary infection of tsetse flies (?)	Short salivary trypanosomes — Inoculative	Man	Game animals (?)	Short blood trypanosomes — Ingestive

With regard to the other forms of sleeping sickness due to trypano

somes these are the transmission agents which carry on the life-cycle of the parasite in *Lamprolaima megistus* (*Triatoma megista*) when

II *Costa with at least one pale area —*

I Mesothorax without true scales

(a) Wing scales mixed dark and light—*Pataganius*

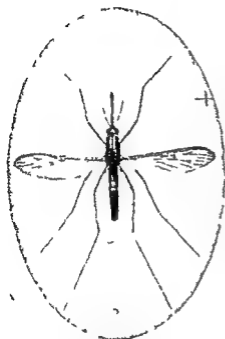


FIG 463 — *Anopheles maculipennis*  
MEIGEN 1818 A CARRIER OF THE  
MALARIAL GERMS

(From a photograph by J J Bell)

James distinguishes between *true scales* which are broad and have distinct striations and *false scales* which are hair like and have indistinct striations

The question whether any of these serve as *definitive reservoirs* by infection of the ova as suggested by Schaudinn has never been proved but we doubt whether much research has been attempted in this direction

The anopheline mosquitoes known definitely to transmit malaria arranged according to recent methods of classification and following Hindle and our previous lists, are as follows —

I Anophelines definitely known to be able to carry the malarial parasites through the complete cycle of quartan tertian, or subtertian infections or only as far as zygotes —

(a) ANOPHELES CARRIERS

Number	Mosquito	Observer	Observation	Habitat
1	<i>A. algeriensis</i> Theobald 1903	The Sergents	To sporozoites	North Africa
2	<i>A. bifurcatus</i> Linnaeus, 1758	Grassi	Tertian	England
3	<i>A. maculipennis</i> Meigen 1818	Many	Quartan tertian malignant tertian	Europe

## TROPICAL KALA AZAR

Parasite	Intermediate Host	Intermediate Reservoir	Transmission	Definitive Host	Method of Infection
<i>Leishmania donovani</i>	Man	(?)	Parasites passed in faeces — Ingestive (?)	Water insect (?) Biting insects (?)	Cysts in drinking water (?) — Ingestive (?) Inoculative (?)

## MEDITERRANEAN KALA AZAR

Parasite	Intermediate Host	Intermediate Reservoir	Transmission	Host Nature Doubtful	Method of Infection
<i>Leishmania infantum</i>	Man	Dogs (?)	Blood sucking insect (?) — Inoculative (?)	Fleas (?)	Blood sucking insect (?) — Inoculative (?) Contaminative (?)

**Coccidiosis** — The discovery of a coccidial oocyst in a fly's intestine by Wenyon and O Connor suggests that possibly this is the method of infection of man by these parasites the fly only acting as an **h**aps this ought to

parasites have as their **definitive host** various species of anopheline mosquitoes. The classification of the Anophelinae is as follows —

- A Costa with less than four main dark spots—*Protoanopheles*
- B Costa with four main dark spots —
  - I Sixth vein with not more than three dark spots—*Drueteroanopheles*
  - II Sixth vein with more than three dark spots—*Neoanopheles*

The *Neoanopheles* have no malarial carriers

## DIVISION I PROTOANOPHELES CHRISTOPHERS 1911

The division contains the following genera —

- A *Costa without pale areas* —
  - I Female palps with second segment disproportionately long—*Stethomyia*
  - II Female palps with second segment not disproportionately long—*Anopheles*



## (e) MYZOMYIA CARRIERS

Number	Mosquito	Observer	Observation	Habitat
8	<i>M albirostris</i> Theobald	Staunton	Malignant tertian zygote	Malaysia
9	<i>M culicifacies</i> Giles	Stephens and Christophers	All forms	India and Ceylon
10	<i>M formosensis</i> I and II Tsuguki = <i>M aconita</i> Dönitz= <i>Anopheles</i> <i>hochii</i> Donitz	Tsuguki	Malignant tertian	Formosa
11	<i>M funesta</i> Giles= <i>M kumassi</i> Chalmers	Many includ ing Chalmers in Kumassi	Malignant tertian including Kumassi zygote and sporozoites	Tropical Africa
12	<i>M hispaniola</i> Theobald	Sergents	Tertian	North Africa South Spain
13	<i>M listoni</i> Liston	Kimoshita Stephens and Christophers	Tertian	India
14	<i>M turkhanii</i> Liston	Stephens and Christophers	Malignant tertian	India

## (f) PYRETOPHORUS CARRIERS

Number	Mosquito	Observer	Observation	Habitat
15	<i>P costalis</i> Loew	Many	All forms	Tropical Africa
16	<i>P myzomyfacies</i> Theobald	Sergeant	Sporozoites	Algeria
17	<i>P superpictus</i> Grassi	Grassi Pignani and Bastienelli	Tertian	Europe

## (g) PSEUDOMYZOMYIA CARRIER

Number	Mosquito	Observer	Observation	Habitat
18	<i>P ludlowi</i>	Christophers	Malignant tertian	Malaysia

## (b) PATAGIAMYIA CARRIER

Number	Mosquito	Observer	Observation	Habitat
4	<i>P. pseudopunctis pennis</i> Theobald	Darling	Malignant tertian	Panama Canal Zone

## (c) CYCLOLEPPTERON CARRIERS

Number	Mosquito	Observer	Observation	Habitat
5	<i>C. mediopunctatum</i> Theobald	Cruz	Tertian	Brazil
6	<i>C. nototrichum</i> = <i>A. intermedius</i> Chagas	Cruz	Tertian	Brazil

## (d) ARRIBALZAGIA CARRIER

Number	Mosquito	Observer	Observation	Habitat
7	<i>A. pseudo maculipes</i> Chagas	Cruz	Tertian	Brazil

## DIVISION II DEUTEROANOPHELES CHRISTOPHERS 1911

This division may be classified as follows —

- A Terminal segment of female palpi less than half length of penultimate  
Tarsi not broadly banded —
  - I Mesothorax without true scales—*Myzomyia*
  - II Mesothorax with true scales—*Pyretophorus*
- B Terminal segment of female palpi at least half length of penultimate  
Tarsi broadly banded —
  - I Mesothorax not completely covered with true scales—*Pseudomyzomyia*
  - II Thorax completely covered with true scales —
    - (a) Abdomen without lateral scale tufts —
      - 1 Palpi moderately shaggy—*Nyssorhynchus*
      - 2 Palpi markedly shaggy—*Myzorhynchella*
    - (b) Abdomen with lateral scale tufts—*Cellia*

Most of these genera possess malarial carriers

II. Anophelines believed to be malarial carriers for epidemiological reasons—

Number	Mosquito.	Observer	Habitat
31	<i>Stethomyia aithens</i> James.	Daniels, Christophers	Malaysia, India
32	<i>Zyretophorus chaudiery</i> Theobald.	Billet	Algerian Oases (saline waters)
33	<i>Myzomyia d'halis</i> Patton	Patton	Aden
34	<i>Myzomyia kochi</i> Dönitz = <i>M aconista</i> = <i>M formosensis</i>	Daniels	Malaysia
35	<i>Myzomyia lutz</i> Theobald	Lutz	Brazil
36	<i>Nyssorhynchus karwari</i> James and Luson	Staunton	Malaysia, India
37	<i>Anopheles (?) martens</i> Laveran	Laveran	Cambodia.
38	<i>Myzorhynchus maurisanius</i> Grandpré (? = <i>M paludis</i> = <i>M aconista</i> )	Ross	Mauritius, Madagascar
39	<i>Anopheles (?) pearsali</i> Laveran	Laveran	Cambodia.

III. Malarial carriers in Lists I and II under synonym names (The ble

(a) .. .. . 100  
(b) .. .. .

(c)

(d)

IV. Probably not carriers:—

(a) *Myzorhynchus barbirostris* Van der Walp India, Ceylon, Malaysia,  
China

(b) .. .. . Ceylon, India, Ceylon, China

V.

America

(h) NYSSORHYNCHUS CARRIERS

Number	Mosquito	Observer	Observation	Habitat
19	<i>N annulipes</i> Walker	Kumoshita	Malignant tertian	Australia
20	<i>N fuliginosus</i> Giles	Stephens Christophers and Addie	Quartan malignant tertian	India
21	<i>N maculatus</i> Theobald	Staunton	Malignant tertian	India
22	<i>N maculipalpis</i> var <i>indianus</i> Theobald	Stephens and Christophers	Malignant tertian	India
23	<i>N stephensi</i> Liston = <i>Neocellia</i> <i>stephensi</i> Theobald	Stephens Christophers Liston and Bentley	Tertian	India
24	<i>N theobaldi</i> Giles	Stephens and Christophers	Quartan malignant tertian	India.
25	<i>N willmors</i> James	Addie	Sporozoites	India

(i) CELLIA CARRIERS

Number	Mosquito	Observer	Observation	Habitat
26	<i>C albimana</i> Weidemann	Darling	All forms	Central and Tropical America
27	<i>C argyrolarvis</i> Desvoidy	Darling	Malignant tertian	West Indies South America
28	<i>C pharacensis</i> Theobald	Newstead Dutton and Todd	Tertian	Egypt
29	<i>C tarsimaculata</i> Goeldi	Darling	Tertian malignant tertian	South America

(j) MYZORHYNCHHELLA CARRIER

Number	Mosquito	Observer	Observation	Habitat
30	<i>M arabiensis</i>	Patton	Sporozoites	Aden Hunterland

- A Gonadial and reno-pericardial cavities communicate—*Isopleura Siphonopoda*  
 B Gonadial and reno-pericardial cavities separate—*Prorhipidoglossomorpha*

The *Isopleura* contains the 'Chitons' and the *Siphonopoda* the *Cephalopods* with which we are not concerned

#### Grade Prorhipidoglossomorpha Grobben

**Definition.**—Mollusca in which the gonadial and reno-pericardial cavities are separate the foot is wholly posterior to the head and a visceral commissure is present

**Classification.**—The Prorhipidoglossomorpha are divided into three classes as follows —

A *Body bilaterally symmetrical* —

I Mantle united ventrally to form a tube / No ctenidia—Class I *Scaphopoda* Bronn

II Mantle not so united ctenidia present—Class II *Lamellisbranchia* de Blainville

B *Body asymmetrical*—Class III *Gastropoda* Cuvier

We are only concerned with the third class

#### Class Gastropoda Cuvier

**Definition.**—Prorhipidoglossomorpha with asymmetrical organization with well developed head with shell formed in one piece and spirally coiled at least in the larva

**Classification** —The class may be divided into two subclasses —

A Visceral commissure twisted into a figure of eight mostly dioecious—*Streptoneura*

B Visceral commissure not so twisted with shortened visceral commissure monoecious—*Euthyneura*

#### Subclass I Streptoneura Spengel

**Definition**—Gastropoda dioecious with a few aberrant genera maximum torsion of visceral mass and commissure / Head with only one pair of tentacles

**Classification**—The *Streptoneura* are divided into two orders —

A Nervous system not concentrated Infracesophageal commissure present Ctenidia bipectinate and free at their distal ends—*Aspidobranchia*

B Nervous system somewhat concentrated Infracesophageal commissure present Ctenidium monopectinate and attached to mantle along its whole length—*Pectinibranchia*

Only the last concerns us

#### Order Pectinibranchia

**Definition**—*Streptoneura* as defined above

**Classification.**—The *Pectinibranchia* are divided into two suborders —

A Without proboscis pallial siphon or Leiblich's unpaired cesophageal poison gland—*Tænioglossa*

B With proboscis pallial siphon and Leiblich's poison gland—*Stenoglossa*

Only the *Tænioglossa* are of importance to us

#### Suborder Tænioglossa

**Definition.**—*Pectinibranchia* with the characters given above with three teeth one lateral and two marginals on each side of the median tooth of the radula

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## MALARIA

Parasites	Definitive Hosts	Definitive Reservoir	Infection	Intermediate Host	Intermediate Reservoir	Transmission
<i>Plasmodium malariae</i> , <i>Plasmodium vivax</i> , <i>Laverania malariae</i>	Anopheline mosquitoes	Unknown	Salivary sporozites — Inoculative	Man	Human carriers	Macrogametocytes and microgametocytes from blood — Ingestive

## B. HELMINTHIASIS.

**Trematode Infections.**—The trematode infections of man have as their intermediate host a mollusc. The definitive host is a vertebrate, from whom the eggs escape in the urine, the feces, or the respiratory secretions. These eggs hatch in water, producing a ciliated, actinopharyngeate miracidium, which enters and

is too heavily infected

It is therefore necessary to note the classification and method of recognition of the known carriers

## PHYLUM MOLLUSCA Cuvier

**Synonyms**—*Palisata* Latreille, *Malacozoa* de Blainville

**Definition.**—Metazoa with no sign of primitive segmentation with well

always present

**Classification.**—The phylum so defined is divided into three grades as follows—

## Subclass II Euthynsura Spengel

Synonym D

radula possessing uniform teeth usually with two pairs of tentacles

- A Marine forms with aquatic respiration—*Opisthobranchiata*  
 B Aerial or fresh water (exceptionally marine) forms with pallial cavity but no ctenidium No free larval form—*Pulmonata*

## Order Pulmonata Cuvier

This order is divided into two suborders—

- A Aquatic forms with a single pair of tentacles—*Basommatophora*  
 B Terrestrial forms with two pairs of tentacles—*Stylommatophora*

## Suborder Basommatophora

Definition.—Pulmonata with an external shell and a single pair of well developed contractile but not invaginable tentacles at the bases of which lie the eyes

Classification—There are some eleven families but only the following concern us—

- A Shell thin dextral no inferior pallial lobe—*Limnæidæ*  
 B Shell sinistral inferior pallial lobe prominent—*Planorbidæ*

## Family Limnæidæ Broderip

Name " " " " " "

Type Genus—*Limnæa* LinnæusGenus *Limnæa* Lamarck

Definition.—*Limnæidæ* with spiral shell more or less elongated thin pointed spire translucent body whorl large aperture rounded columella obliquely twisted

Eu  
D

*uncatula* Muller  
Isles *L. viator*

## Family Planorbidæ Adams

Definition.—*Basommatophora* with sinistrally coiled shell Inferior pallial lobe very prominent transformed into a branchia tentacles tapering

Classification—

- A Shell discoid branchia not folded—*Planorbis*  
 B Shell ovoid with prominent spire Branchia not folded—*Bulinus*

~~Classification~~

### Tribe Platypoda

**Definition**—*Tænioglossa* with the characters given above

**Classification**.—There are some fifty five families of which we are interested in the *Melaniidæ* only

### Family Melaniidæ Gray

**Definition**.—*Platypoda* with spiral shell and elongated spire  
Operculum horny Foot short Mantle border fringed Viviparous Fluvatile

**Classification**—Several genera

A Shell long—

I Shell turriculated aperture enlarged anteriorly—*Melania*

II Spiral very long, aperture notched anteriorly—*Faunus*

B Shell short, thick, and aperture rounded—*Other genera*

### *Melania* Lamarck

**Synonyms**—*Thiara* Mergele, *Pyrgula* Crist

**Definition**—*Melaniidæ* with turricated shell acute apex, whorls ornamented with striæ or spires, aperture oval, pointed above, outer lip sharp sinuous Operculum subspiral

**Type Species**—*Melania hastula* Leach

**Remarks**—Some 400 or more species distributed throughout Southern Europe India, the Philippines Japan and the Pacific Islands Distinct groups in the S.thern U. S. A.



FIG 464—*Blandfordia contractus* (p 893)



FIG  
180  
OF  
Jap

### *Blandfordia* Adams 1863

**Definition**—*Melaniidæ* with ovate conical shell, apex truncated Aperture elliptical peristome continuous operculum subspiral Rostrum elongated Tentacles very short, eyes sessile Foot large, divided into two

We are only concerned with *B japonica* which is the carrier of *Schistosomum japonicum*



## Family Physidæ Dall

th visceral mass and shell  
a narrow aperture, tentacles

end

*Physa* or *Physopsis africanus* is the carrier of *S. hamatobium* in Brazil

## TREMATODE INFECTIONS

Trematode	Definitive Host	Definitive Reservoir	Transmission	Intermediate Host	Infection
<i>Fasciola hepatica</i>	Man	Sheep	Miracidium in water — Penetrative	Species of Læmnaea	Cercaria encysted on weeds — Ingestive
<i>Fasciolopsis buski</i>	Man	Pig	Miracidia in water	Shrimps (?)	(?) — Ingestive (?)
<i>Metagonimus yokogawai</i>	Man	Unknown	Ditto	<i>Melania libertina</i>	Cercaria under scales of fish — Ingestive
<i>Laragonimus ingeri</i>	Man	Carriera	Ditto	Ditto	Cercaria in water — Penetrative or in crabs ingestive
<i>Clonorchis sinensis</i>	Man	Cats oogs pigs	Ditto	Ditto	Cercaria encysted in muscles of fish — Ingestive
<i>Schistosoma hamatobium</i>	Man	Man	Ditto	Subgenera and species of <i>Bulinus</i>	Cercaria in water — Penetrative
<i>Schistosoma mansoni</i>	Man	Man	Ditto	Species of <i>Planorbis</i>	Cercaria in water — Penetrative
<i>Schistosoma japonicum</i>	Man	Cats	Ditto	<i>Blandfordia nosophora</i> vel <i>japonica</i>	Cercaria in water — Penetrative

Genus *Planorbis* Guettard

**Definition**—As above. Shell discoidal dextral many whorled aperture crescentic Peristome thin incomplete upper margin projects

**Type**—*Planorbis corneus* Linnaeus

*Planorbis bolssyi* Potiez and Michaud 1838

**Synonym**—*P. laurenti* Bourguignat

**Remarks**—This is the intermediate host of *Schistosoma mansoni* in Egypt and the Sudan as discovered by Leiper

*Planorbis olivaceus* Spix

This is the carrier of *S. mansoni* in Brazil



FIG 466—*Planorbis olivaceus* THE CARRIER OF *Schistosoma mansoni* IN BRAZIL

Genus *Bulinus* Adamson

**Synonyms**—*Nautia* Leach *Aplexa* Fleming Often spelt *Bullinus*

**Definition**—Planorbidae with ovoid shell prominent spire, branchia folded

**Type**—*Bulinus hypnorum* Linnaeus

**Classification**—The species of importance to us are—*Bulinus contortus* Michaud 1829 *Bulinus dybowski* Fischer 1891, *Bulinus alexandrina* Innes *Bulinus innesi* Bourguignat

The above are the hosts of *Schistosoma haematobium* in Egypt

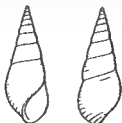


FIG 467—*Physa* (*Physopsis*) *africana* BRAUSS A CARRIER OF *Schistosoma haematobium*

## NEMATODE INFECTIONS

(These are on the same lines as trematode and cestode as a rule)

Nematode	Definitive Host	Definitive Reservoir	Infection	Intermediate Host	Transmission
<i>Filaria bancrofti</i>	Man	Man	Microfilaria in blood — Ingestive	Culex and Stegomyia	Larvofilaria — Penetrative
<i>Loa loa</i>	Man	Goat (?) sheep (?)	Microfilaria — Ingestive	Species of chrysops	(?)
<i>Dracunculus medinensis</i>	Man	Man	Larvæ in water — Ingestive	Species of cyclops	Water — Ingestive
<i>Ascaris lumbricoides</i>	Man	Pig	Eggs — Ingestive	Rats	Larvæ on food — Ingestive

it has deposited its larvæ on the skin of sheep and become a producer of myiasis thereon

Myiasis will form the subject matter of Chapter LXVII (p 1619) and here it is only necessary to say that the larvæ may be deposited

there is a curious observation, which is that an *intermediary animal carrier* exists, at all events, in the case of *Dermatobia cyaniventris*, which has been studied by Blanchard, Surcouf, Rincones Tovar, Zepeda, and Sambon

According to the last named observer, *D. cyaniventris* lives in Trinidad and Central and South America and its young produce cutaneous myiasis in man, monkeys (the brown howler and the capuchin), pumas, agoutis, cattle goats, pigs, and birds (the toucan

## CESTODE INFECTIONS

(These are on the same lines as the trematode infections)

<i>Cestode</i>	<i>Definitive Host</i>	<i>Definitive Reservoir</i>	<i>Infection</i>	<i>Intermediate Hosts</i>	<i>Transmission</i>
<i>Dibothrio cephalus latus</i>	Man	Dogs cats	Eggs in water	Cyclops fish	Plerocercoid in muscles — Ingestive
<i>Dipylidium caninum</i>	Man	Dogs cats	Eggs on fur	Dog and cat louse or flea	Dog licking cysticerci Cat milk cysticerci — Ingestive
<i>Hymenolepis diminuta</i>	Man	Rats mice	Eggs on fur	Meal broth beetles rat flea	Food cysticerci — Ingestive
<i>Tænia solium</i>	Man	Man	Eggs in faeces	Pigs	Cysticerci in muscles — Ingestive
<i>Tænia saginata</i>	Man	Man	Eggs in faeces	Cattle	Cysticerci in muscles — Ingestive
<i>Echinococcus</i>	Dog jackal	Dog jackal	Eggs on fur	Man sheep	Cysticerci in muscles — Ingestive

## II MYIASIS.

Myiasis comprises the infestation of the vertebrate body with the *larvæ of diptera* and the disorders which arise in the body of the host therefrom

The vertebrate is an *intermediate host* because not merely is it

human food cooked and raw: such as milk meat butter sweets etc and the distances within reason to which flies travel is limited by the necessities of food and shelter but they go to the nearest

In Africa flies belonging to the genus *Pycnosoma*—e.g. *P. marginale*—do the same as do other species in India and China

These flies one and all are great feeders and are accustomed to vomit frequently while they pass a considerable amount of excrement Graham Smith records 1102 vomit marks and nine faecal deposits on an area of a cupboard window 6 inches square Bearing this in mind it can readily be appreciated how well they contaminate food and what efficient disseminators of germs they may be

Graham Smith describes faecal deposits as round opaque often raised spots of a yellowish brownish or whitish colour while vomit spots have an opaque centre and a clear periphery bounded by a darker zone

But the body of the fly is thickly clothed with hairs or setae and as it walks over filth particles containing bacteria are apt to cling to these hairs

We therefore have to consider —

- A The external carriage of germs
- B The internal carriage of germs

With regard to the former Graham Smith's experiments with *B. prodigiosus* show that this bacillus can be cultivated from the legs and wings of infected flies for eighteen hours after infection It must be remembered that flies are everlastingly cleaning themselves and it is a matter of common knowledge how the proboscis is rubbed by the anterior pair of legs which become contaminated therefrom

Therefore the external carriage of germs from filth to food is possible provided that it takes place within a relatively short time

With regard to the internal carriage the same observer has shown that though there is no evidence that *B. prodigiosus* multiplies therein it can live in the alimentary canal of flies for four to five days

The investigation of the presence of *B. typhosus* in a fly is most difficult because there are non lactose fermenters present as normal denizens of the fly Faichnie has shown that it tends to be present in the intestine and not on the legs but Cochrane's experiments show that it may be recovered from the external washings of flies The bacilli so obtained were tested with typhoid serum and gave positive reactions and even by immunizing animals therewith and testing the serum so obtained against stock *B. typhosus* positive results were obtained

The history is as follows —

This much is known but we may well ask whether this happens with any other insects Sambon says that the common house fly

Restricting our attention to *Dermatobia cyaniventris* the chart of its carriage is as follows —

#### DERMATOBIA MYIASIS

Parasite	Intermediate Hosts	Infection	Intermediate Host	Intermediate Reservoir	Transmission	Definitive Stage
Larva of <i>Dermatobia cyaniventris</i>	<i>Jaïnsonia</i> Carries eggs Only female	Mosquito sucks blood larva enters wound — Penetrative	Man	Warm blooded vertebrates	Larva escapes from skin	Free living

#### D BACTERIAL DISEASES

The spores of bacteria enable them to spread from host to host with a degree of protection during the passage but non sporing forms will be benefited by the aid of a carrier which not merely affords protection but also a means of dissemination We will divide the discussion into flies fleas and lice

##### I FLIES

This intermediary host is often a non blood sucking fly of the nature of the common house fly which is a potential carrier of disease because it and its kind frequent decaying matter and excreta for the purpose of laying the eggs while both it and its larvæ are filth feeders

If it and its allies only fed upon filth there would be but little harm but unfortunately, they are attracted to many articles of

Weeks bacillus and a spreader of the severe forms of ophthalmia found therein

Flies can also carry the plague bacillus in living virulent form in their alimentary canal for forty-eight hours, but they do not play any great part in the dissemination of the disease

bacteria, and as such are worthy of study

We require to know the house flies of the tropics but these are little investigated though Nicholls at St Lucia has found the following breeding in human faeces —

*Drosophila melanogaster* Meigen  
*Limosina punctipennis* Wiedemann  
*Sepsis* species  
*Sarcophaga aurifinis* Walker  
*Sarcophaga* species  
*Sarcophagula* species

In Africa and the East generally —

*Pycnosoma marginale* Wiedemann  
*Pycnosoma chloropyga* Wiedemann

In India by Patton —

*Musca domestica* Linnæus  
*Musca domestica* var *determinata* Walker  
*Musca nebulosa* Fabricius  
*Musca pattoni* Austen

In England the investigation is much more complete—e.g.—

<i>Musca domestica</i> Linnæus	<i>Fannia scalaris</i> Fabricius
<i>Musca corvina</i> Fabricius	<i>Anthomyia radicum</i>
<i>Calliphora erythrocephala</i> Meigen	<i>Sarcophaga carnaria</i> Linnæus
<i>Calliphora vomitoria</i> Linnæus	<i>Sepsis punctum</i> Meigen
<i>Lucilia cæser</i> Linnæus	<i>Prophila casei</i> Linnæus
<i>Pollenia rudis</i> Fabricius	<i>Scatophaga stercoraria</i> Linnæus
<i>Fannia canicularis</i> Linnæus	<i>Drosophila fenestrarum</i>

Also *Scenopinus fenestralis* and species of *Stomoxys* and of *Psychoda* but these two last have already been described and now it behoves us to look at the classification and structure of the non biting flies

We have already given the classification of the Diptera in Chapter XXXIII, and need only consider that of the families

#### FAMILY PHORIDÆ

*Phora femorata* occurs occasionally in houses *Aphiochæta ferruginea* Brunner causes intestinal myiasis

#### FAMILY SCENOPINIDÆ

*Scenopinus fenestralis* Linnæus is the so called window fly which is probably the only household fly which is not injurious to health

With regard to the spread of *B typhosus* and *B paratyphosus* A and B via the larva to the fly, the only experiments of real moment are those of Faichne, who worked with uncultivated germs, the flies being bred in infected faeces. He showed that this was highly probable, though other workers have failed with cultivated material. Faichne, however, did not say that he separated the larvæ which had fed upon the excrement therefrom and therefore did not say that he had excluded the possibility of the newly hatched flies feeding upon the excrement. Hence the subject of the carriage from the larva to the imago is *sub judice* at present.

importance

For epidemiological reasons supported by bacteriology, it appears

intestine for twelve or more days, and thus can contaminate food by the faeces up to the fifth day, and sometimes up to the sixth to fourteenth day.

Anthrax spores remain infective in flies for twenty days, being found in the faeces, while in dead flies the period is indefinite, moreover, they can pass via the larva to the imago.

At this stage we may point out that the infection of wounds produced by biting flies—  
biting flies Patton wa

be remembered

it is possible that it may occur as the means of infection of unusual sites, as Graham Smith has shown that the germ can live in the crop and intestine for twenty four hours and longer, and, further, that the vital germs—



- II Hind metatarsi not incrassate and always longer than the following joint
- (a) Discal and basal cells united anal cell absent front bare or at most bristly above small usually light coloured flies—*Oscinidæ*
- (f) " " " " " " " " " " " "

Oral vibrissæ present Arista long plumose—*Drosophilidæ*

#### FAMILY SEPSIDÆ

The Sepsidæ include the genus *Prophila* of which the species *Prophila casca* Linneus may cause intestinal myiasis *Sepsis violacea* is a dung fly often found in houses

#### FAMILY OSCINIDÆ Latreille 1804

*Muscoidea acalyptata* with front without bristles crown with only a few short bristles border of mouth without vibrissæ Middle tibia with small spurs costa of wings without bristle Subcostal vein absent Anterior of two small basal cells united with discal cell posterior wanting

The genera with which we are concerned are—*Microneurum* Becker 1903 and *Hippelates* Loew 1863

#### *Microneurum* Becker 1903

#### *Microneurum funicola* de Meijere 1905

e - - - - -

vib.)  
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ed to  
this It chiefly attacks the eyelids and the ears and since the experiments performed by Sir Allan Perry and Castellani it has been suspected as a possible carrier of the Koch Weeks bacillus which is the cause of severe attacks of conjunctivitis It objects to the odour of such substances as Odol which may be used to drive it away

#### *Hippelates* Loew 1863

Some authorities consider this genus not well founded and state that a revision of the genera of the Oscinidæ is urgently required

The species *Hippelates flabipes* Loew *H. plebejus* Loew and *H. pusio* Loew attack men and domestic animals by darting at the eyes and other parts of the body in search of moisture or perspiration They also attack wounds sores scratches ulcers Their life history is unknown In habits they are diurnal frequenting open and sunny places They are found in the southern United

## FAMILY IMPIDÆ

of the face & legs with medium or small bodies and small heads

ceous

It is doubtful whether these insects attack man. As a rule they live on the juices of other insects and plants

## SUBORDER II. CYCLORRHAPHA.

**Section 1: Aschiza.**—This group includes the family Syrphidæ, of which no species is known to bite man

**Section 2: Schizophora.**—This group includes the true flies characterized by a distinct frontal lunula and a frontal suture, antennæ with three simple segments, and an arista which is generally dorsal. They may be classified into—

## Muscoidea.

without squamæ covering the

**TRIBE 2 Muscoidea calyptrata,** with squamæ covering the halteres

## MUSCOIDEA ACALYPTRATÆ

A large number of families are grouped together under this division, of which the most important for our purposes are—

- 1 Sepsidæ
- 2 Oscinidæ
- 3 Drosophilidæ
- 4 Borboridæ

These families can be recognized as follows—

- A Subcostal (auxiliary) vein present. Radial 1 (first longitudinal) terminates near or beyond the middle of the wing
  - I With a distinct bristle on each side of the face near the oral margin. Front never bristly near antennæ. abdomen somewhat elongate cylindrical, usually narrowed near base. Small black flies found about decaying matter—*Sepsidæ*
  - II Subcostal vein absent vestigial or incomplete. Radial 1 usually ends in the costa before the middle of the wing. Head not produced into lateral processes
    - I Hind metatarsi incrassate and usually shorter than the second joint—*Borboridæ*

Remarks—It has been known for many years that flies can

proved that flies were capable of transmitting cholera. In the Spanish American War in the Army Concentration Camps of 1898 flies were found to be spreaders of typhoid. Lime was sprinkled on the fecal pits and the flies on the soldiers' mess table were noticed to have their legs whitened with the lime.



FIG 469—HEAD OF *Musca domestica*  
LINNÆUS FEMALE  
(After C J Martin)  
ch Pseudo tracheæ



FIG 470—LEG OF *Musca domestica*  
(After C J Martin)

The flies which breed in human excrement in America are (1) *Musca domestica* house fly (2) *Drosophila ampelophila* fruit fly (3) *Fannia canicularis* little house fly (4) *F. brevis* little house-fly (5) *Stomoxys calcitrans* stable fly (6) *Plora femorata* (7) *Sarcophaga tritialis*

Of these the most common are the house and the stable flies. The house fly

In the Boe of typhoid of Soil Fabr

tion was published by Furth and Horrocks in the *British Medical Journal* and they showed that the ordinary house-fly (*Musca domestica*) can convey enteric infective matter from excreta or polluted materials or objects on which they may walk rest or

protection, and eucalyptus oil or a menthol spirit lotion can be sprinkled on the coat collar to drive them away.

#### FAMILY DROSOPHILIDÆ.

This family includes the species *Drosophila ampelophila* Loew, commonly called the fruit-fly, and found hovering over fruit in houses, and as it is attracted by excrementum it must be regarded as dangerous. There are a large number of species of *Drosophila* known.

#### FAMILY BORBORIDÆ

This family includes *Borborus equinus*, a small fly sometimes found in houses.

### MUSCOIDEA CALYPTRATÆ

#### MUSCINÆ

*Musca* Linnaeus, 1761

*Musca domestica* Linnaeus, 1761

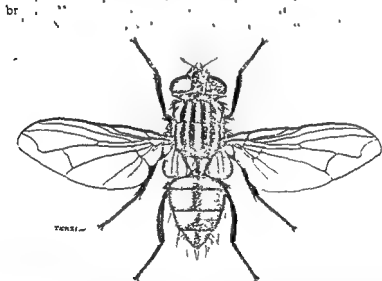


FIG. 468.—*Musca domestica* LINNAEUS (×6)

Scutellum grey, with black sides. Wings tinged pale grey with yellowish base. Legs blackish-brown. Abdomen yellowish, with dorsal blackish brown line, except the last segment, which is entirely blackish-brown. Eyes separated by a wide frontal stripe in the female, and near together in the male. Length, 6 to 7 millimetres, wing breadth, 13 to 15 millimetres.

Bacot and Martens

in  
 bac  
 to

... it into close relationship with man, and because the curve of its epizootic begins to rise ten to fourteen days before that of the epidemic. This period is calculated to be made up of three days, during which the flea leaves the dead rat to which is added an incubation period of four days. This is the

This rat flea will not merely bite man, but is capable of living for some time on man's blood, and is often found on human beings after inspection of plague-stricken houses.

Further, it is believed that the spread of plague is due, not to migration of rats, but to the carriage of infected rats on ships, and of fleas in merchandise or on human beings. The Commission apparently consider the last to be the most important method.

Pneumonic plague, which occurs only in 25 per cent of cases during bubonic epidemics, spreads from man to man by bacilli carried by the air, for Strong and Teague demonstrated that the sputum in pneumonic plague frequently contains bacilli which have shown

unless there is a considerable amount of aqueous vapour in the atmosphere, as is found in very cold climates, and hence the tendency for pneumonic plague to spread in those rather than in warm climates. On the other hand the bubonic or septicæmic is not spread from man to man, but from rats to man. The epizootic is the real disease, and the epidemic is only an offshoot.

The above ætiology explains fully the predisposing causes of sex, age, occupation, season, of house, of season, of climate, and also the carriage of the disease from one place to another by people, fodder, grain, bales of cotton and clothing, rags, etc.

... having found 22 per cent infected with *B. pestis* when collected from infected native huts. Moreover, he successfully transmitted the disease from man to the rat by means of *C. rotundatus*.

In California, Wherry, McCay, and others have shown that the ground squirrel (*Citellus beecheyi*) is subject to plague, and that its commonest flea, *Ceratophyllus acutus* Baker, is the vector from

The flies found by Newstead in houses in Liverpool or bred from refuse and excreta were *Musca domestica* in quite 90 per cent of all flies while the other species met with were—*Calliphora erythrocephala* the blow fly *Scatophaga stercoraria* the dung fly *Borborus equinus* a small fly *Stomoxys calcitrans* the stable fly *Fannia canicularis*, *F. scalaris* *Anthomyia radicum* the root fly *Psychoda phalaenoides* the owl midge

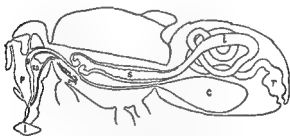


FIG 471—ANATOMY OF *Musca domestica*  
(After C J Martin)

L Labella P proboscis SD near salivary duct g gullet S mid gut  
C sucking stomach T rectum

- 1 Fore feet modified for digging, their claws very large, eyes  
and ears very small from the front but very large

2

sometimes scaly,

- (c) With large external cheek pouches—*Heteromyida*  
 (d) Without external cheek pouches —  
 (1) Hind feet not greatly elongated, little if any longer  
 than front feet, tail not longer than rest of body  
 —*Murida*  
 (2) Hind feet greatly elongated much longer than  
 front feet, tail much longer than rest of body—  
 —*Zapodida*

II Fur thickly sprinkled with sharp stiff spine-like quills—*Erethizontida*

- B Upper front teeth four the second pair minute and placed directly behind  
 the first pair, hind legs much longer than front legs, ears very large,  
 tail very short (suborder *Duplicidentata*)—*Leporida*

### FAMILY SCIURIDÆ Gray, 1821

#### *Squirrels and Marmots.*

The genera of the *Sciuridæ*, which concern us, may be recognized by the  
 following table taken from Swenk —

- A Sides without a furred membrane (subfamily *Sciurinae* true squirrels and  
 marmots)

I Tail long much over one-half of length of body, form slender,  
 coloration usually spotted or streaked

(a) Cheek pouches absent, tail bushy the hairs growing outward  
 arboreal (squirrels)—*Sciurus*

(b) Cheek pouches present large, tail well haired but not bushy  
 mainly terrestrial

I Nail of thumb well developed, back conspicuously striped  
 lengthwise with five dark and two or four white  
 stripes

(A) Premolars in upper jaw one on each side, back with two  
 white stripes, rump rufous, tail with hairs shorter  
 than rest of body (eastern chipmunks)—*Tamias*

II

- (a) always plain  
 (b) always plain

- II Sides with a densely furred membrane joining front and hind legs (sub-  
 family *Pteromyinae* flying squirrels)—*Sciuopterus*.

squirrel to squirrel, and, further, that this flea will bite man. Further, they record a subacute case of plague in a boy where the infection was believed to be acquired by contact with ground squirrels. With regard to the outbreak in Manchuria and North

and sleeping together in overcrowded insanitary inns, especially as the cold of the winter induced an indoor existence. These

(*Spermophilus citellus*), which is common around Mukden was susceptible to the infection. There has been an epizootic in Suffolk, and a few cases of bubonic plague in man.

We therefore have to consider the rôle of the flea and the rôle of

is necessary to say a few words with regard to the rats

## CLASS MAMMALIA.

### SUBCLASS EUTHERIA

#### ORDER GLIRES LINNÆUS 1758

**Definition**—Euthera with toes armed with claws. Size usually small or medium. Front teeth chisel shaped and separated from the grinding teeth by a wide space.

**Classification**.—This classification is taken from Swenk—

A Upper front teeth two, both large (suborder *Simplidentata*)

1 *Fur not sprinkled with quills*

(a) Tail very broad flat scaled, hind feet webbed, size large—  
*Castoridae*

(b) Tail round or compressed, hind feet not webbed, size small to medium



rufescent brown on the dorsum white or grey on the venter but may be darker or lighter. Mammae two or three.



FIG 474—RAT PLAGUE RESERVOIR

There are two varieties of the rat *alexandrinus* which is large and heavier and *rufescens* which is smaller and redder but there are any number of intermediate species. It breeds frequently throughout the year.

It is essentially a house rat, living in the tiles or thatch of the roof or in holes and recesses of the floor but it will live in the crevices of coconut trees.

Its pathogenicity is important for it is the plague rat of Upper India. It is supposed to have entered Europe with the Asiatic invasion.

*Epimys norvegicus* Erxleben 1777

Synonym — *Mus decumanus* Pallas 1778

Large heavy rats with heavily and uniformly tapering tails, dark coloured dorsally, lighter coloured ventrally, only 80 per cent of the length of the head and body. Heavy flesh coloured feet, short round ears and broad heavy snout.

Colour brown on the dorsum and dirty white on the venter. Foot pads large, heart shaped, mammae ten to twelve in number.



FIG 475—HEAD OF *Epimys rattus*  
(After Hossack)

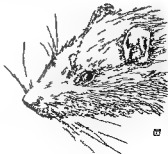


FIG 476—HEAD OF *Epimys norvegicus*  
(After Hossack)

Genus *Citellus* Oken 1816

Synonym — *Spermophilus* F. Cuvier 1825 *Citellus* Lichtenstein

Definition.—As above

Type Species — *Citellus citellus* Linnaeus 1766

We are however concerned with *C. beecheyi* and with *M. bobak*

*Citellus beecheyi* Richard 1829

Definition.—*Citellus* of large size with large prominent ears Tail more than two thirds of body length Colour above mixed black and pale yellowish brown

Remarks — This is the ground squirrel of California mentioned above

Genus *Marmotta* Blumenbach 1779

Synonym — *Arctomys* Storr, 1780

Definition — As above

Type Species — *Marmotta marmorata* Linnaeus 1758

*Marmotta bobak* Muller 1776

Definition.—*Marmotta* of medium size resembling the type but of uniform colour being above yellowish brown overlaid with black

Remarks — This is the tarbagan of Mongolia and Central Asia

## FAMILY MURIDÆ Gray 1821

*Rats and Mice*

The genera of the Muridæ which concern us may be recognized as follows —

A Crowns of grinding teeth with tubercles arranged in transverse rows

I Rows of tubercles in grinding teeth three very distinct in upper jaw tail long and scaly Subfamily *Murinae* (Old World rats and mice)

II Rows of tubercles in grinding teeth two tail generally hairy Subfamily *Cricetinae* (American rats and mice)

B Crow

I

II

Subfamily *Murinae* Blaud 1857

Definition — As above

Remarks — The *Murinae* comprise the genus *Epimys* Trouessart 1857 to which belong the rats and mice of which two are of great importance in the spread of plague—viz *Epimys rattus* and *E. norvegicus*

Genus *Epimys* Trouessart 1857

Definition.—*Murinae* with first and second upper molars with two tubercles on inner side Upper incisors with outer cutting edge entire

*Epimys rattus* Linnaeus 1758

Synonym.—*Mus rattus* Linnaeus

Slender rats with very pointed muzzles and large out standing ears large prominent eyes long tail, and greyish black fur

They therefore behave more like the bacteria than like animal parasites in the carrier

For our present purpose relapsing fevers may be divided into two groups as follows —

I The *Louse Group*, which is characterized by being carried by the louse. These are the European, the North African, the Indian and the American types

From the infective blood the spirochætes pass into the alimentary canal of the louse and from thence into its coelom where they remain and from there they find their way into the eggs, which are infective, as are their larvæ

Infection of man is brought about by contaminative means — the louse in biting causes irritation, the man scratches his skin causing abrasions, and at the same time kills a louse, crushes it and rubs it into the abrasions, which become infected. Nicolle and Blazot believe that the organisms which are infective are granules just before they reappear as spirochætes. Sergeant and Foley have stated that there were very small virulent forms in man during apyrexia

In this life cycle there is no definitive host, merely two hosts of equal value. Only the insect is the *preservative host* and the vertebrate the *intermediary host*

### I THE LOUSE GROUP OF RELAPSING FEVERS

Parasites	Preservative Host	Preservative Reservoir	Infection	Intermediary Host	Transmission
<i>S. recurrentis</i> <i>S. berbera</i> <i>S. carteri</i> <i>S. novyi</i>	Pediculus corporis	Lice by hereditary transmission	Small forms in body cavity — Contaminative	Man	Small blood forms — Ingestive

**Spirochætal Epidemic Jaundice** — This is caused by *Spiroschaudinnia icterohæmorrhagica*. The reservoir in rats and after living in the blood.

It is believed to have been caused but very rarely as the result of a rat bite. It is introduced here as a convenient place though not known to be due to lice

Parasite	Host	Reservoir	Method of Infection
<i>Spiroschaudinnia icterohæmorrhagica</i>	Man	Rats (?)	Contaminated water (?)

This is the brown sewer or ship rat which is supposed to have come from China to Europe and from Europe to India. It is the plague rat of Bombay. It is very prolific, producing several litters of eight to ten young per annum.

The chart of plague is as follows —

## PLAGUE

Organism	Infected Host	Infected Reservoir	Transmission	Propagative Host	Infection
<i>Bacillus pestis</i>	Man	Murine	Bacilli obtained by blood-sucking — Ingestive	Fleas	Bacilli in faeces into wound by bite — Contaminative More rarely inoculative

## 3 LICE

occur with *Bacillus cuenoti* in the germ cells of the cockroach



FIG 477—*Pediculus corporis* DE GEER 1775 MALE CARRIER OF TYPHUS ETC



FIG 478—*Pediculus corporis* DE GEER 1778 FEMALE CARRIER OF TYPHUS ETC

(From a photograph by J J Bell)

## E DISEASES OF UNKNOWN CAUSATION.

The evidence in favour of pellagra being an insect borne disease has not increased and many authorities believe it to be a deficiency disorder. The insects which were accused were species of *Simulium* and some Chironomid biting flies. There are suggestions that Rocky Mountain fever and tsutsugamushi disease are bacterial infections and Noguchi has found a spirochæte in yellow fever.

Disease	Host	Reservoir	Transmission	Carrier	Infection
Pappataci fever	Man	—	Blood sucking — Ingestive	Species of phlebotomus	Bites — Inoculative
Dengue fever	Man	—	Blood sucking — Ingestive	<i>Stegomyia culex</i> (?)	Bites — Inoculative
Yellow fever	Man	Man in mild attacks — Monkeys (?)	Blood sucking — Ingestive	<i>Stegomyia calopus</i>	Bites — Inoculative
Rocky Mountain fever	Man	—	Blood sucking — Ingestive	<i>Dermacentor venustus</i> <i>D. andersoni</i>	Bites — (?)
Tsutsugamushi disease	Man	<i>Arvicola natanae sunni</i> (?)	Blood sucking — Ingestive	<i>Microtrombidium akamushi</i>	Bites — (?)
French fever spirochæte (?)	Man	—	Blood sucking — Ingestive	Lice	Bites — Contaminative
Typhus	Man	—	Blood sucking — Ingestive	Lice	Bites — (?)
Acute anterior poliomyelitis	Man	—	Blood sucking — Ingestive	<i>Stomoxys calcitrans</i>	Bites — Inoculative

## F. CHANCE TRANSMISSION.

Numerous blood sucking and non blood sucking insects may by chance obtain an organism and carry it in their proboscis and directly infect an open sore—e.g. flies and jaws which according to Castellani's observations and experiments is far from a rare occurrence.

II The *Tick Group* which is characterized by being spread by the genus *Ornithodoros*. The known forms are the African (perhaps a separate East African) the Persian the Colombian with the Panamanian.

This group differs from the louse group in the pre-eminence of the *infective granule* which according to many authorities is in itself doubtful.

Spirochaetes enter the tick with the infective feed bore their way into the cells of various organs and break up into coccoid bodies the infective granules. These granules pass into the second generation and so the tick is a *preservative reservoir*. They also pass into the Malpighian tubules from which they escape when the thick white Malpighian excrement is passed which only takes place towards the end of a feed. The spirochaetes now enter the new host via the hole made in the skin by the tick for its meal of blood.

## II THE TICK GROUP OF RELAPSING FEVERS

<i>Parasite</i>	<i>Preservative Host</i>	<i>Preservative Reservoir</i>	<i>Infection</i>	<i>Intermediate Host</i>	<i>Transmission</i>
Forms allied to <i>S. duttoni</i> and found in Africa East Africa Colombia Panama and perhaps in Persia	Species of <i>Ornithodoros</i> — e.g. <i>O. moubata</i> <i>O. turicata</i> <i>O. talaga</i> and perhaps <i>O. savignyi</i>	The ticks by hereditary transmission	The infective granules(?) — Contaminative	Man	Blood spirochaetes — Ingestive

We have followed Balfour's suggestion that the Persian relapsing fever is not caused by *Argas persicus* but by a species of *Ornithodoros* probably *O. savignyi*. It must be admitted that many authorities disbelieve in the infective granule.

## 4 PHLEBOTOMUS FLIES

*Verruga Peruviana*—It has been suggested that the carrier of this disease may be *Phlebotomus verrucarum* Townsend 1913 but this appears to be very doubtful.

## SECTION II VEGETAL PARASITES

### CHAPTER XXXVI SCHIZOMYCETES

Preliminary—Thallophyta—Schizomycetaceae—Eubacteriales—Coccaceae—  
Bacillaceae—Spirillaceae—Mycobacteriaceae—References

#### PRELIMINARY

In our previous editions we drew attention to the numerous text books and easily available works on bacteriology which we decided not to consider and we hold to that view still but we notice that the works on this subject as supplied to the student of medicine are perhaps somewhat lacking in systematic classification

To meet this need we have written the present chapter which merely considers those bacteria which are of importance from the point of view of tropical medicine and instead of giving descriptions of their characters these are merely indicated by tables Hence

In the meanwhile the reader can find the existing rules in *Règles Internationales de la Nomenclature Botanique* published in Jena

## G IMPERFECT CARRIAGE OF PARASITES

Parasites may develop up to a certain point in the alimentary canal of insects in which they are unable to complete their life cycle—e.g. the malarial germ in many anophelines only proceeds as far as the zygote

## H TERMS

## TERMS FOR ANIMAL PARASITES

<i>Definitive host</i>	Host with sexual life of parasites
<i>Intermediate host</i>	Host with asexual life of parasites
<i>Intermediary host</i>	No development of parasite. Merely carriage
<i>Definitive reservoir</i>	Natural supply of infection of intermediate host
<i>Intermediate reservoir</i>	Natural supply of infection for definitive host
<i>Transmission</i>	Passage from intermediate to definitive host
<i>Infection</i>	Passage from definitive to intermediate host
<i>Contaminative</i>	Abrasions or bites contaminated by faeces etc
<i>Inoculative</i>	Parasites injected by insect during biting
<i>Ingestive</i>	Parasites taken into alimentary canal <i>per os</i>

## TERMS FOR BACTERIA

<i>Infected host</i>	Vertebrate infected by bacterium
<i>Infected reservoir</i>	Vertebrate carrier
<i>Protective host</i>	Arthropod carrier without germ increasing in number
<i>Propagative host</i>	Arthropod carrier with germ increasing in number
<i>Transmission</i>	As in animal parasites
<i>Infection</i>	

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SECTION II  
VEGETAL PARASITES

CHAPTER XXXVI  
SCHIZOMYCETES

Preliminary—Thallophyta—Schizomycetacea—Eubacteriales—Coccaceae—  
Bacillaceae—Spirillaceae—Mycobacteriaceae—References

PRELIMINARY.

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in 1912, and we may perhaps be permitted to remind him that names of orders should end in *-ales*, or suborders in *-ineae* and of families <sup>règles</sup> <sup>Jena</sup>

The *Regnum Vegetabile* or vegetal kingdom is usually divided into four great phyla or groups—viz the Thallophyta the Briophyta the Pteridophyta and the Phanerogamæ but of all these only the first need concern us

The Thallophyta include a great variety of plants whose vegetative body may consist of one or many cells forming a more or less branched structure

These plants may be defined and classified as follows —

**PHYLUM THALLOPHYTA**

Definition — Vegetabilia thallophyta

- A Thallophyta with chromatophores and often with chlorophyll—  
Class I *Algae* Roth 1797
- B Thallophyta without chromatophores or chlorophyll—Class II  
*Fungaceæ* Linnæus 1757

There can be no doubt that the Fungaceæ are descendants of the Algae which because of a saprophytic or parasitic environment have altered their food habits and have adapted themselves to new methods of nutrition and hence no longer require chromatophores or chlorophyll as they no longer manufacture their food with the  
vegetal material

which is usually

*ce tersa*

visions probably

evolution thus the *Schizomycetes* are probably

etes the Phæo-

## SCHIZOMYCETACEA Naegeli 1857

**Definition**—Thallophyta without chlorophyll and as a rule without chromatophores with the vegetative body consisting of a single cell in which the nucleus is not present in the form typical for other thallophytes. Reproduction by fission or spore formation.

**Classification**—The Schizomycetes may be divided into orders as follows—

- |   |                    |                            |
|---|--------------------|----------------------------|
| A | Cocci              | Eubacterales               |
| B |                    |                            |
| C |                    | a gelatinous<br>III Micro- |
|   | <i>bacteriales</i> |                            |

Only the first order contains forms of importance in tropical medicine.

### ORDER I EUBACTERIALES

**Definition**—Schizomycetes which contain neither sulphur nor bacterio purpurein.

**Classification**—The Eubacteriales may be divided into families as follows—

- A Cells in free condition usually globular in division somewhat elliptical—Family 1 *Coccaceæ* Zopf 1885 *emendavit* Migula 1900
- B Cells long or short cylindrical straight division one direction—Family 2 *Bacillaceæ* Fischer 1894
- C Cells spirally curved or representing part of a spiral division in one direction
- D ( )
- E ( )

The Chlamydobacteriaceæ do not concern us but the other families require some consideration.

#### FAMILY COCCACEÆ Zopf 1885 *emendavit* Migula 1900

- Gamma ( ) usually
- Alpha ( ) 1877
- Beta ( ) forms of importance
- A P ( )

#### B F

or zoogloea masses and often Gram negative and when pigmented usually yellow or red—Tribe II *Micrococceæ* Trevisan 1880 *emendavit* Winslow and Rogers 1905

we wrote on this subject in regard to the Protozoa hold good here and need not be repeated while we would refer the reader again to the section on evolution in Chapter V p 117

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The Thallophyta include a great variety of plants whose vegetative body may consist of one or many cells forming a more or less branched structure

These plants may be defined and classified as follows —

**PHYLUM THALLOPHYTA**

n =

- A Thallophyta with chromatophores and often with chlorophyll—  
Class I *Algæ* Roth 1797
- B Thallophyta without chromatophores or chlorophyll—Class II  
*Fungicæ* Linnæus 1737

The center of the plant is the

or chlorophyll as they no longer manufacture their food with the aid of sunlight but subsist on decaying animal or vegetal material

the Peridineæ the Conjugatæ the Diatomenæ the Heterocontæ the Chlorophyceæ the Characeæ the Phycomycetes the Phæophyceæ the Rhodophyceæ the Eumycetes

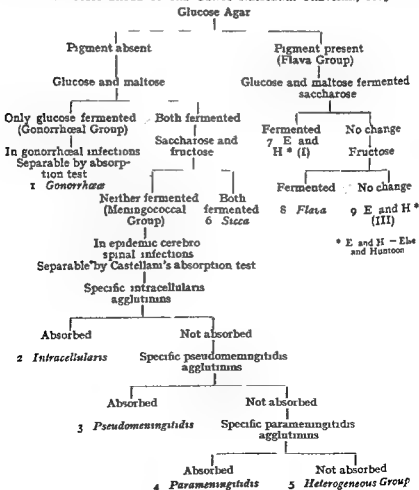
We will now turn to consider the Schizomycetes

**Classification.**—The genus *Neisseria* includes the following species

7 *A number of heterogeneous strains*, separable by the agglutination and absorption tests of specific immune sera

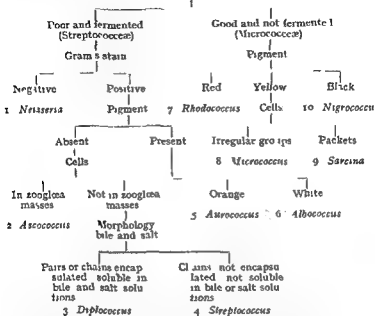
These various strains and groups can be differentiated from one another by the tests set forth in the following diagnostic table —

DIAGNOSTIC TABLE OF THE GENUS *NEISSERIA* TREVISAN, 1885



The two tribes may be divided into genera as set forth in the following table —

DIAGNOSTIC TABLE OF THE FAMILY COCCACEÆ ZOFF 1885  
Growth and Carbohydrates



We are however only concerned with certain genera of the Streptococceæ which are *Neisseria*, *Diplococcus*, *Streptococcus*, *Aurrococcus* and one genus of the Micrococceæ—viz, *Nigrococcus*. As *Micrococcus meliensis* is often elongated (coccobacillus), we propose to place it under a separate heading *Incertæ Sedis*.

TRIBE 1 STREPTOCOCCEÆ TREVISAN 1889 emendat WINSLOW AND ROGERS 1905

Genus *Neisseria* Trevisan 1885

Synonyms—*Micrococcus* Hallier 1866 *pro parte*, *Diplococcus*



Genus *Diplococcus* Weichselbaum, 1886, *emendatus* Winslow  
and Rogers, 1905

pneumonia, of which there are many strains separable by serum reactions. The genus requires further study before a definite

Genus *Streptococcus* Rosenbach, 1884, *emendatus* Winslow  
and Rogers, 1905

tropics, as primary agents causing the pathological changes, and as secondary or terminal infections in the course of illnesses due to other causes.

They are responsible for several types of puerperal fever, of abscesses, of septicæmias and of skin diseases.

**Natural Habitat.**—Most streptococci are found as parasites (or saprophytes) of the nose, nasopharynx, and alimentary canal of vertebrate animals, but they can also be found in the alimentary tract of the invertebrata, and apparently also, but to a less extent, living on or in plants.

From these natural homes they may pass to air, soil, milk, or water, and so may be conveyed from one animal to another, as they

in normal  
faeces and  
of native

in normal faeces and of native equine and bovine faeces

Researches with regard to certain human passages have shown how quickly these become infected with streptococci after birth



It may be that *S. mitior* Schottmuller 1903 is the same as *mitis* Andrewes and Horder 1906. It has been found in case of puerperal fever.

### IX FÆCALIS GROUP

**Definition**—Streptococcus parasitic in man facultative anaerobic growing in broth and upon agar and blood serum without the formation of pigment and well upon gelatine at 22° C without production of liquefaction, fermenting glucose saccharose lactose mannitol

quantitatively

**Remarks**—This is an important pathogenic group in the tropics being found in all sorts of conditions. Its various strains may be differentiated as follows—

- A Glucose saccharose lactose salicin and mannitol fermented  
 Typical subgroup  
 I Raffinose not fermented—*S. faecalis* Andrewes and Horder 1906  
 II Raffinose fermented—*S. versatilis* Broadhurst 1915
- B Suppression of one of the characters of the typical subgroup or with the addition of the fermentation of inulin—Atypical subgroup  
 I Raffinose not fermented—Variants of *S. faecalis*  
 II Raffinose fermented—Variants of *S. versatilis*

### X SALIVARIUS GROUP

**Definition**—Streptococcus parasitic in animals facultative anaerobic without pigment or gas formation growing in broth and upon agar at 22° C in gelatine which is not liquefied fermenting glucose saccharose lactose mannitol and raffinose may ferment inulin but not mannitol and generally incapable of clotting milk. Habitat human saliva human faeces bovine and equine faeces

**Classification**—This group can be divided into a typical subgroup and varieties of the typical subgroup. The important characters are—

**TYPICAL SUBGROUP**—Glucose saccharose lactose and raffinose and sometimes inulin fermented

- A Salicin not fermented—  
 I Haemolysis marked—*S. anginosus*  
 II Haemolysis absent—*S. salivarius*
- B Salicin fermented—  
 I Haemolysis marked—*S. actuosus*  
 II Haemolysis absent—*S. bovinus*

**ATYPICAL SUBGROUP**—Contains varieties of *S. anginosus*, *S. salivarius*, *S. actuosus* and *S. bovinus* in which there is suppression of some characters but as these suppressions are generally only temporary it is not necessary to specially characterize them.

**Remarks**—These streptococci are frequently found in sore throats and other infections in the tropics.

mented

A Raffinose and inulin not fermented and neutral red not reduced—*Typical subgroup*

I Saccharose fermented—*S. equinus*

II Saccharose not fermented—Andrewes and Horder Winslow and Palmer Broadhurst (A) Fuller and Armstrong

B Raffinose or inulin fermented or neutral red reduced—*Atypical subgroup*

We have found *S. equinus* Andrewes and Horder, 1906, in a case of septicæmia in the tropics

### VIII. ERYSIPELATUS GROUP

**Definition.**—Streptococcus parasitic in animals, aerobic facultative anaerobe, without pigment, does not liquefy gelatine or produce gas, ferments lactose, glucose, saccharose and salicin, but not mannitol or raffinose

**Classification.**—The group may be differentiated into strains as follows —

A Colonies on agar large white and opaque at the end of forty-eight hours resembling those of an albococcus, milk clotted Hæmolytic action unknown—*S. puerperalis*

B Colonies on agar small, translucent, not resembling at the end of forty-eight hours those of an albococcus —

I Hæmolytic present,—

(a) Milk clotted —

1 Some kind of a capsule present colonies on agar typical —*S. epidemicus*

2 Capsule absent growth on agar may be absent or in the form of very fine or at times watery colonies but typical on acidic agar—*S. equi*

(b) Milk not clotted —

Capsule absent colonies on agar typical —

1 Found in cases of erysipelis—*S. erysipelatos*

2 Found in dermatitis cupuliformis—*S. tropicalis*

II Hæmolytic absent —

(a) Milk clotted—*S. mitior*

(b) Milk not clotted—*S. mitis*

which Chalmers and O Farrell in 1913 named and more fully described. It is found in *Trichomycosis rubra* and we differentiate the three species as follows —

- A Does not ferment glucose—*Castellani*
- B Produces slight acidity in glucose —
  - I Nitrates reduced to nitrites—*Roseus*
  - II Nitrates not reduced—*Fulvus*

### Genus *Nigrocooccus* Castellani and Chalmers, 1918

Definition.—Micrococceæ saprophytic rarely parasitic, producing

with *Cohnistreptothrix tenuis* Castellani (1911) other forms are *N. fuscus* Adamez (1888) in water *N. cyaneus* Schroeter, 1870, in air and water, and they may be differentiated as follows —

- A Gelatine liquefied—*Fuscus*
- B Gelatine not liquefied —
  - I Pigment indigo blue—*Cyaneus*
  - II Pigment black—*Nigrescens*

### INCERTÆ SEDIS

The so called *Micrococcus melitensis* Bruce 1886 which is the causal agent in Mediterranean Malta or undulant fever is difficult to classify, because elongated forms are seen at times in cultures and because it seems to have no affinity with the Gram negative cocci, but, on the other hand is very like the typhoid-colon group of organisms in certain respects. It does not ferment sugars nor produce indol does not liquefy gelatine nor show polar staining while milk becomes alkaline.

### FAMILY BACILLACEÆ Fischer 1894

Definition.—Eubacteriales with cells long or short flagellate or non flagellate sporogenous or non sporogenous but always cylindrical and straight. They divide in one direction only.

Type Genus.—*Bacillus* Cohn, 1872

Remarks.—The enormous numbers of species and varieties gathered together under the names *Bacterium* and *Bacillus* form such an unwieldy mass that we have endeavoured to simplify matters by formulating a number of tribes with genera.

Classification.—The family Bacillaceæ may be classified into tribes as follows —

Growth in ordinary laboratory media —

- A Entirely or almost entirely absent—Tribe 1 *Nitrobacteræ*
- B Poor Gram negative grow best on blood media—Tribe 2 *Hæmophilæ*
- C Extremely slow and scanty growth on ordinary and blood media—Tribe 3 *Graciloidæ*
- D Growth good —
  - I Endospores present—Tribe 4 *Bacillæ*
  - II Endospores absent —

SHOTTMULLER'S CLASSIFICATION OF THE GENUS STREPTOCOCCUS—This is very simple and is based on the characters of colonies on blood agar plates

1. Colonies surrounded by a clear zone of hæmolytic—*S. hæmolyticus*  
of a peculiar  
medium (cocci)

### Genus *Aurococcus* Winslow and Rogers 1905

defined as follows —

Remarks—This group is of great interest in the tropics as its  
us parts—e.g.

ion showed it  
g and Wherry  
h seems to be

### TRIBE II MICROCOCCÆ TRIVISAN 1889 *emendat* WINSLOW AND ROGERS 1905

#### Genus *Rhodococcus* Winslow and Rogers 1905

Definition—Micrococceæ usually saprophytes rarely parasites with cells in groups or regular packets. Generally more or less decolorized by Gram. Growth on agar abundant with the formation of red pigment. Very slight fermentative action. Gelatin rarely liquefied.

1905  
to the  
groups to

await further investigation

We add *Rhodococcus castellani* discovered by Castellani and

Of all these the most important from our present point of view is *Eberthia* which contains many intestinal organisms

TRIBE ENCAPSULATEÆ CASTELLANI AND CHALMERS 1918

**Definition**—Bacillaceæ growing well on ordinary laboratory media without endospores, neither fluorescent nor chromogenic aerobes not liquefying gelatine possessing capsules in animal tissues

**Type Genus**—*Encapsulatus* Castellani and Chalmers 1918

Genus *Encapsulatus* Castellani and Chalmers 1918

**Definition**—Encapsulateæ with the tribal characters

capsulated pleomorphic organisms which ferment glucose and

A

B

II Inositol fermented with the formation of acid and gas—*Lactis aerogenes*

TRIBE EBERTHIEÆ CASTELLANI AND CHALMERS 1918

**Definition**—Bacillaceæ growing well on ordinary laboratory media not forming endospores aerobes and often facultative anaerobes liquefying gelatine and possessing capsules

h may

A Glucose and lactose either not at all or only partially fermented with the production of acid but no gas —

I Milk not clotted —

(a) Glucose and lactose not fermented—Genus 1 *Alcaligenes* Castellani and Chalmers 1918

(b) Glucose partially fermented with the production of acid and no gas lactose not fermented —

1 Motile—Genus 2 *Eberthia* Castellani and Chalmers 1918

2 Non motile—Genus 3 *Shigella* Castellani and Chalmers 1918

(c) Lactose and glucose partially fermented with the production of acid but no gas—Genus 4 *Dysenteroides* Castellani and Chalmers 1918

(a)  
(b)

## THE TRIBES

Tribe	Type Genus	Type Species	Original Name of Type Species
Nitrobacteriæ	<i>Nitrobacterium</i> Castellani and Chalmers 1918	<i>Nitrobacterium</i> <i>nitrobacter</i> (Winogradsky 1892)	<i>Nitrobacter</i> Winogradsky 1892
Hæmophilæ	<i>Hæmophilus</i> Castellani and Chalmers 1918	<i>Hæmophilus</i> <i>influenzae</i> (Pfeiffer 1892)	<i>Bacillus</i> of <i>influenza</i> Pfeiffer 1892
Graciloidæ	<i>Graciloides</i> Castellani	<i>Graciloides</i> <i>albifaciens</i>	<i>Bacillus albifaciens</i> Castellani 1904
Bacilliæ	<i>Bacillus</i> Cohn 1872 <i>pro parte</i>	<i>Bacillus subtilis</i> (Ehrenberg 1833)	<i>Vibrio subtilis</i> Ehrenberg 1833
Bacterioidæ	<i>Bacteroidium</i> Schroeter 1872	<i>Bacterium</i> <i>prodigiosum</i> (Ehrenberg 1838)	<i>Monas prodigiosa</i> Ehrenberg 1838
Bacteroidæ	<i>Bacteroides</i> Castellani and Chalmers 1918	<i>Bacteroides</i> <i>fragilis</i> Veillon and Zuber	<i>Bacillus fragilis</i> Veillon and Zuber
Proteæ	<i>Proteus</i> Hauser 1885	<i>Proteus vulgaris</i> Hauser 1885	<i>Proteus vulgaris</i> Hauser 1885
Pasteurellæ	<i>Pasteurella</i> Tom and Trevisan 1889	<i>Pasteurella gallinae</i> Tom and Trevisan 1889	Microbe du cholera des Indes Pasteur 1887
Fberthæ	<i>Fberthus</i> Castellani and Chalmers 1918	<i>Fberthus typhosus</i> Zopf 1845	<i>Bacillus</i> of Fberth auctores
Encapsulatae	<i>Encapsulatus</i> Castellani and Chalmers 1918	<i>Encapsulatus</i> <i>pneumoniae</i> (Friedländer 1883)	<i>Pneumococcus</i> The micrococcus of <i>pneumonia</i> Friedländer 1883

**Genus Alcaligenes Castellani and Chalmers 1918**

**Definition**—Ebertheæ which do not ferment glucose or lactose and are characterized by their general lack of fermentative power and by actually increasing the alkalinity of the media. Milk is not clotted and is rendered alkaline.

**Type**—*Alcaligenes faecalis* (Petruschky 1896) *emendatus* Castellani and Chalmers 1918

Here also comes *Alcaligenes vivax* (Archibald 1918) which was

**Classification**—These various organisms can be differentiated as follows —

- A Non motile—*Metalkaligenes*
- B Motile —
  - I No acidity in any sugar—*Faecalis*
  - II Acidity in mannitol—*Vivax*

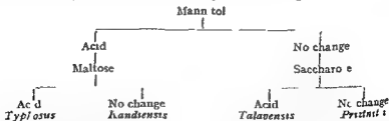
**Genus Eberthus Castellani and Chalmers 1918**

**Definition**—Bacillaceæ motile partially fermenting glucose with the production of acid and no gas. Lactose not fermented. Milk not clotted.

as well as a number of species which are the causal agents of forms of enteroidæ.

**Classification**—The genus contains the following species in addition to the type—*E. kandianensis* Castellani, *E. talavensis* Castellani, *E. pristinii* Castellani.

They may be differentiated biochemically as follows though they can be distinguished in addition by their serological reactions —

**Genus Shigella Castellani and Chalmers 1918**

**Definition**—Ebertheæ non motile partially fermenting glucose with the production of acid but no gas. Lactose not fermented. Milk not clotted.

**Types**—*Shigella dysenteriae* (Kruse 1899)

**Remarks**—This genus includes a number of forms which are associated with bacillary dysentery but in going through those which have been described we have rejected all with very imperfect characters which all never permit of their recognition.

## II Milk clotted —

Glucose partially fermented with the production of acid but no  
acid and gas.—Genus 5

no gas:—

I. Milk not clotted—Genus 8, *Wesenbergus* Castellani and Chalmers,  
1918

D Glucose and lactose completely fermented with the production of  
acid and gas.—

I Milk not clotted—Genus 9, *Enteroides* Castellani and Chalmers,  
1918.

II. Milk clotted—Genus 10 *Escherichia* Castellani and Chalmers  
1918

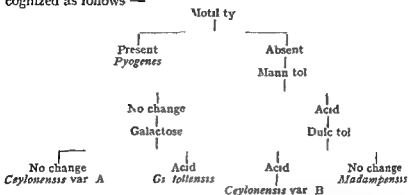
In order to be quite definite, we give the following table showing  
the type species for each genus —

Genus	Type Species	Original Name of the Type Species
Alcaligenes	<i>Alcaligenes faecalis</i> (Petruschky, 1896)	<i>Bacillus faecalis</i> <i>alkaligenes</i> Petruschky, 1896
Eberthus	<i>Eberthus typhosus</i> (Zopf, 1885)	<i>Bacillus</i> of Eberth auctores
Shigella	<i>Shigella dysenteriae</i> (Kruse, 1899)	<i>Bacillus dysenteriae</i> Kruse, 1899
Lankoides	<i>Lankoides pyogenes</i> (Passet, 1902)	<i>Bacillus pyogenes</i> <i>faecalis</i> Passet, 1902
Dysenteroides	<i>Dysenteroides meladysentericus</i> (Castellani, 1917)	<i>Bacillus meladysentericus</i> Castellani, 1917
Salmonella	<i>Salmonella paratypha</i> (Schottmüller, 1902)	<i>Bacillus paratyphosus</i> A Schottmüller, 1902
Balkanella	<i>Balkanella coagulans</i> (Castellani, 1916)	<i>Bacillus coagulans</i> Castellani, 1916
Wesenbergus.	<i>Wesenbergus wesenbergi</i> (Castellani, 1913)	<i>Bacillus wesenbergi</i> Castellani, 1913
Enteroides	<i>Enteroides entericus</i> (Castellani, 1907)	<i>Bacillus entericus</i> Castellani, 1907
Escherichia	<i>Escherichia coli</i> (Escherich, 1886)	<i>Bacterium coli commune</i> Escherich, 1886

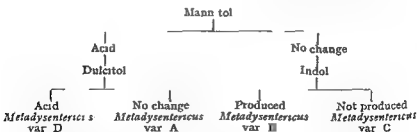


Genus *Lankoides* Castellani and Chalmers 1918

**Definition**—Ebertheæ fermenting glucose partially with the production of acid but no gas lactose not fermented or only partially  
 P  
 i  
 nus may be recognized as follows —

Genus *Dysenteroides* Castellani and Chalmers 1918

**Definition**—Ebertheæ fermenting glucose and lactose partially  
 Milk not clotted  
*sentericus* (Castellani 1917)  
 ie organisms of the Meta  
 dysenteric group which may be differentiated as follows —

Genus *Salmonella* Lignieres *emendavit* Castellani and Chalmers 1918

**Definition**—Ebertheæ which completely ferment glucose but do not ferment lactose and partially or completely ferment mannitol

Classification —The species belonging to this genus may be divided for purposes of recognition into —

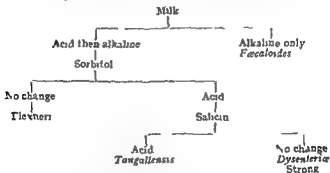
*Flexnerella* (Flexner group sensu lato)

The forms belonging to these divisions and sections may be recognized by the following tables —

Subgenus *Flexnerella* Castellani and Chalmers 1918

(MANNITOL PARTIAL FERMENTERS)

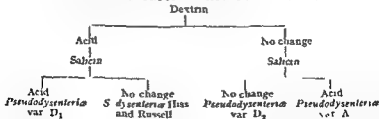
FLEXNER GROUP MALTOSÉ PARTIAL FERMENTERS



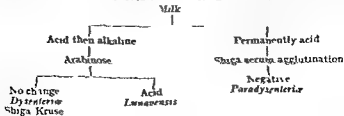
Subgenus *Flexnerella* Castellani and Chalmers 1918

(MANNITOL PARTIAL FERMENTERS)

PSEUDODYSENTERY GROUP MALTOSÉ NON FERMENTERS



SHIGA KRUSE DIVISION MANNITOL NOT FERMENTED



## ASIATICUS DIVISION INDOL FORMERS

Saccharose

No change

Acid and gas

Voges Proskauer

Salicin

Positive

Negative

Acid and gas

No change

*Archibaldi* Castellani  
and Chalmers*Carolina*  
Castellani*Pseudo asiatica*  
Castellani

Motility

Present  
*Asiatica* var *mobilis*  
CastellaniAbsent  
*Asiatica*  
CastellaniGenus *Balkanella* Castellani and Chalmers, 1918*noises* Castellani—which may be separated as follows—

Saccharose

No change  
*Coagulans*Acid and gas  
*Carolinoides*Genus *Wesenbergus* Castellani and Chalmers 1918

Definition.—Ebertheæ which ferment glucose completely and

ind pro  
se and  
It is

an indol producer

To the same group belongs *Wesenbergus gumus* Castellani, which is non motile

Archibald in the Anglo-Egyptian Sudan obtained an organism of this type from the blood of a case of enteroidæa in Khartoum on the fifth day of the illness. It was motile formed acid and gas in glucose galactose and rhamnose (iso-dulcitol) dextrin starch mannitol and sorbitol but only acid in lactose levulose maltose and dulcitol, while it failed to ferment saccharose raffinose inulin salicin, glycerol erythrol or adonitol. It did not produce indol

- A Mannitol not fermented—*Morgan group*  
 B Mannitol partially fermented with the production of acid but no gas—*Veboda group*  
 C Mannitol completely fermented with the production of acid and gas—*Paratyphoid Asiaticus group*

The Morgan group only contains *Salmonella morganii* which is the same as Morgan I of older nomenclature

## VEBODA GROUP

This group contains two organisms—viz *Salmonella veboda* Castellani 1909 and *S. willegoda* Castellani 1911 They may be distinguished as follows—



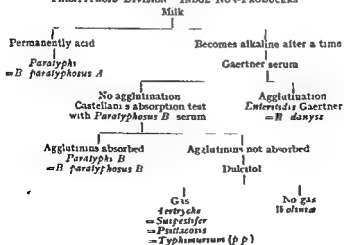
## PARATYPHOID ASIATICUS GROUP

This group contains a number of forms some of which are of tropical importance It may be classified as follows—

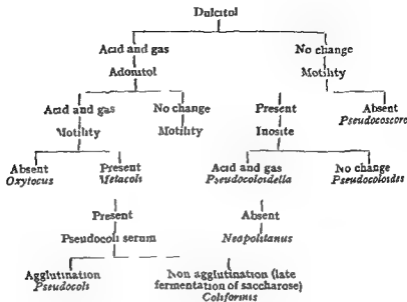
- A Indol not produced—*Paratyphoid division*  
 B Indol produced—*Asiaticus division*

The first subgroup contains *S. paratyphi A*, *S. paratyphi B*, *S. acetylica*, *S. wolinskyi* The term *S. paratyphi C* has been applied by various authors to indicate different germs one of which is identical serologically with *S. enteritidis*

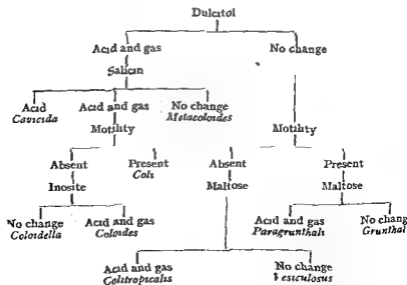
## PARATYPHOID DIVISION INDOL NON-PRODUCERS



SMITH'S INDOL PRODUCING DIVISION  
 Durham's Saccharolytic Communitis Section



SMITH'S INDOL PRODUCING DIVISION  
 Durham's Non Saccharolytic Communitis Section



by Alexander in 1914 in the supplement to the Annual Report of the Local Government Board which may well be classified here

The named species may be separated as follows —

- A. Indol produced —
  - I Dulcitol fermented—*Wesenbergi*
  - II Dulcitol not fermented—*Gaumas*
- B. Indol not produced—*Fermentosus*

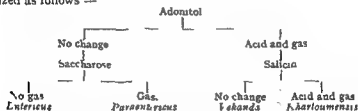
**Genus *Enteroides* Castellani and Chalmers 1918**

**Definition.**—Ebertheæ which ferment glucose and lactose completely with the production of acid and gas Milk not clotted

**Type Species** —*Enteroides entericus* (Castellani, 1907)

of enteroidea in the Anglo Egyptian Sudan

**Classification** —The various species of the genus may be recognized as follows —



**Genus *Escherichia* Castellani and Chalmers 1918**

**Definition** —Ebertheæ which ferment glucose and lactose completely milk clotted

**Type Species** —*Escherichia coli* (Escherich 1846)

**Classification** —The number of species gathered together under this genus even after the rejection of those so imperfectly described that they cannot be classified is so large that they require to be divided into groups and sections as follows —

- A. Indol produced—*Smith's indol division*
  - I Saccharolytic—*Communis* section
  - II Non-saccharolytic—*Communis* section
- B. Indol not produced—*Smith's non-indol division*

We recognize the mistake which has been made with regard to the organism called *Coscoroba* which as originally described belongs to the genus *Pasteurella*—*i.e.* among the hemorrhagic septicæmia being a cause of disease and death in swans. By some mistake years ago quite a different organism belonging to the Coli

ent confusion we pro-  
the name *Escherichia*  
1918 The species may

Bacteria	Motility	Gram	Gelatine	Serum	Lactimus Milk	Lactose	Saccharose	Dulcite	Mannite	Glucose	Maltose	Devrin	Raffinose	Arabinose
<i>acidus lactici</i> uppe	O	O	O	O	AC	AG	O	O	AG	AG	AG	AG	AG	AG
<i>aertryke</i> : De <i>ibele</i>	+	O	O	O	A Alk	O	O	AG	AG	AG	AG	O	O	AG
<i>profaciens</i> Cas ani 1905	O	O	O	O	AC	O	O	O	O	A	O	—	—	—
<i>hibaldi</i> Castel i and Chal 1918	+	O	O	O	A Alk	O	O	AG	AG	AG	AG	—	O	—
<i>stans</i> Cas ani 1905	O	O	O	O	A Alk	O	AG	O	AG	AG	AG	AG	AG	AG
<i>anicus mobilis</i> tellani 1914	+	O	O	O	A Alk	O	AG	O	AG	AG	AG	AG	AG	AG
<i>totensis</i> Cas ani 1912	+	O	O	O	A	A	A	As	O	A	A	O	As	O
<i>apsulatus</i> fler	O	O	O	O	AC	AG	AG	O	AG	AG	AG	AG	AG	AG
<i>rolinus</i> Cas ini	+	O	O	O	A Alk	O	O	O	A or AG	A or AG	A or AG	—	AG	AG
<i>toida</i> Brieger	+	O	O	O	AC	AG	O	AG	AG	AG	O	AG	AG	AG
<i>ylonensis</i> A ellani 1905	O	O	O	O	AC	O	O	O	O	A	O	O	O	O
<i>lonensis</i> B ani, 1905	O	O	O	O	AC	A	A	A	A	A	A	A	A	A
Jordan	+	O	+	+	AC	AG	AG	O	AG	AG	AG	AG	AG	AG

SMITH'S NON INDOL-PRODUCING DIVISION

This division contains only one organism *Escherichia coli mutabilis* Massini, insufficiently described

TRIBE PASTEURELLÆ CASTELLANI AND CHALMERS 1918

Genus *Pasteurella* Toni and Trevisan, 1889

(Trétrop 1900) belongs to this genus as well as *Pasteurella pestis* Kitasato and Yersin 1894. The two great divisions the animal and the human diseases may be separated as follows —

- A No growth on MacConkey's medium containing glucose levulose galactose or mannitol—*Animal group*
- B Growth on MacConkey's medium containing the above mentioned sugars—*Plague*

This test must however be confirmed by animal inoculations

TRIBE PROTEÆ CASTELLANI AND CHALMERS 1918

**Definition**—Bacillaceæ growing well on ordinary laboratory media not forming endospores aerobic without fluorescence or pigment formation but liquefying gelatine

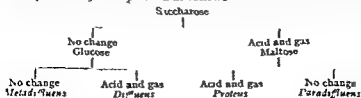
**Type Species**—*Proteus vulgaris* Hauser 1885

**Classification**—The tribe may be divided into genera as follows —

- A Rapid gelatine liquefiers, do not ferment lactose, mostly Gram positive—*Proteus*
- B Slow gelatine liquefiers, ferment lactose Gram negative—*Cloaca*

Chalmers 1918

Classification as the serological reactions are not always in accord with the biochemical characters. To this group belongs *Proteus* No. 19 (see page 1336). Some species may be separated as follows —





Bacteria	Motility	Gram	Gelatine	Serum	Lysin in Nisla	Lactose	Saccharose	Dilute	Mannite	Glucose	Maltose	Dextrin	Raffinose
<i>B. coagulans</i> Castellani	O	O	O	O	AC	O	O	—	O	AG	AG	—	—
<i>B. coli</i> Esche- rich	+	O	O	O	AC	AG	O	AG	AG	AG	AG	AG	AG
<i>B. coli nuda</i> <i>bilis</i> Massini	O	O	O	O	AC	AG	O	O	—	—	—	—	—
<i>B. coloides</i> var <i>A</i> Castellani	O	O	O	O	AC	AG	O	AG	—	AG	AG	—	—
<i>B. coloides</i> var <i>B</i> Castellani	O	O	O	O	AC	AG	O	AG	—	AG	AG	—	—
<i>B. colotropica</i> <i>Its</i> Castel- lani 190	O	O	O	O	AC	AG	O	O	AG	AG	AG	AG	AG
<i>B. colombensis</i> Castellani 1905	+	O	O	O	Avs Alk D or A	O or Gvs	O	AG	AG	AG	AG	As Gs	O
<i>B. coscoroba</i> (= <i>B. pseudo</i> <i>coscoroba</i> ) Castellani and Chal- mers	O	O	O	O	AC	AG	AG	O	AG	AG	AG	AG	AG
<i>B. danysii</i>	—	—	—	—	—	—	—	—	—	—	—	—	—
<i>B. diffluens</i> Castellani 1915	+	O	+	+	Alk D	O	O	O	O or A	AG	O	O	O
<i>B. douglassii</i> Castellani and Chal- mers 1918	O	O	O	O	Alk	O	O	A	A	A	A	—	—
<i>B. dysenteriae</i> Flexner	O	O	O	O	A Alk	O	O	O	A	A	A	A	A
<i>B. dysenteriae</i> Hiss and Russell	O	O	O	O	A Alk	O	O	O	A	A	O	A	A
<i>B. dysenteriae</i> Shiga Kruse	O	O	O	O	A Alk	O	O	O	A	O	O or As	O	C
<i>B. dysenteriae</i> Strong	O	O	O	O	AC	O	A	A	A	A	O	A	A
<i>Shiga</i> Cas- tellani 1911	O	O	O	O	O	AG	O	AG	AG	AG	AG	AGs	OD

Sorbitol	Galactose	Lactose	Inositol	Sucrose	Amygdalin	Tartrate	Erythritol	Glycerol	Indol	Voges Prosk	Broth	Remarks
AG	AG	AG	O	O	-	-	O	-	+	O	Gt	Belongs to the capsula bacilli differs from <i>B. lactis aerogenes</i> in not fermenting inositol differs from <i>B. coli trochalis</i> in being capsulated and in fermenting adonite and not fermenting salicin
AG	AG	AG	AG	O	O	AG	O	A	O or +s	O	Gt	Identical culturally and serologically with <i>B. suspensifer</i> identical culturally with <i>B. terribilis</i> Gaertner (fermentation by agglutination tests) and <i>B. paratyphosus B</i> (no fermentation by Castellani's absorption test agglutination not sufficient)
-	A	A	-	O	-	-	-	-	O	-	-	Very slow and scanty growth on agar
-	AG	-	-	-	-	-	-	-	+	+	Gt	-
AG	AG	AG	O	O	O	AG	O	AG	+s	O	Gt	-
AG	AG	AG	O	O	O	AG	O	AG	+s	O	Gt	Differs from <i>B. asiatica</i> only in being motile
O	A	A	A	As	O	O	O	A	+	O	Gt	-
AG	AG	AG	AG	-	-	-	-	-	±	+	Gt	Capsulated probably identical with <i>B. lactis aerogenes</i>
A	AG	AG	-	-	-	-	-	-	+	O	Gtor + P	-
-	AG	AG	O	A	-	-	-	-	+	O	-	Brieger described it first as non motile differs from <i>B. coli</i> not fermenting maltose
O	O	O	O	O	O	O	O	O	O	O	Gt	-
A	A	A	O	O	O	A	O	A	+	O	Gt	-
AG	AG	AG	A or O	O	-	-	O	-	+	+	Gt	Liquefaction of gelatin very slow The important intestinal liquefying bacilli may be grouped as follows (1) lactose fermenters ( <i>B. cloacae</i> ) (2) lactose non fermenters Gram + ( <i>B. proteus vulgaris</i> ) (3) lactose not fermenters Gram O ( <i>B. dysenteriae</i> )



Sorbitol	Galactose	Lactose	Inositol	Saccharin	Amygdalin	Isodulcitol	Erythritol	Glycerine	Indol	Loges Prosk	Broth	Remarks
AG	AG	—	—	—	—	—	—	—	+	—	—	—
AG	AG	AG	O	AG	O	AG	O	AG	+	O	Gt	—
—	—	—	—	—	—	—	—	—	O	—	—	Incompletely described late lactose fermenter (after six days) said not to produce indol
—	—	—	■	AG	—	—	—	—	—	—	—	—
—	—	—	AG	AG	—	—	—	—	—	—	—	—
AG	AG	AG	O	AG	O	AG	O	AG	+	—	—	Differs from <i>B. coli</i> in being non motile and in non fermenting dulcitol from <i>B. neapolitanus</i> in not fermenting saccharose and dulcitol
AG	AG	AG	O	AG	O	AG	O	AG	+	O	Gt	—
AG	AG	—	A	—	—	—	—	—	O	—	—	Differs from <i>B. coli tropici</i> in fermenting saccharose certain authors use the term <i>B. coscoroba</i> to indicate a different germ with all the characters of the fowl cholera bacillus ( <i>pasteurella</i> )
—	—	—	—	—	—	—	—	—	—	—	—	Culturally and serologically identical with <i>B. enteritidis</i> Gaertner (Bairdbridge)
—	AG	A or AG	—	O	—	—	—	A	O	—	Gt	See remarks on <i>B. cloacae</i> Some strains clot and peptonize milk
—	—	—	—	—	—	—	—	—	+	—	—	—
O	A	A	—	O	O	O	O	O	+	—	—	—
O	A	A	—	O	O	O	O	O	+ or ±	—	—	—
O	A	A	—	O	—	—	As	—	O	—	—	—
A	A	A	—	O	O	A	O	O	+	—	—	—
AG	AG	AG	—	—	—	—	—	—	+	O	Gt 1's	—

Bacteria	Molality	Gram	Gelatin	Serum	Leucine Milk	Lactose	Saccharose	Dulcitate	Mannite	Glucose	Maltose	Dextrin	Raffinose
<i>B. melacolorides</i> Castellani	+	0	0	0	AC	AG	0	G	AG	AG	AG	AG	AG
<i>B. meladiffuens</i> Castellani	+	0	+	+	Alk	0	0	0	0	0	0	0	0
<i>B. meladysen- tericus</i> Cas- tellani 1904 var A	0	0	0	0	A or Alk	A	A	0 or As	A	A	A	—	—
<i>B. meladysen- tericus</i> Cas- tellani 1904 var B	0	0	0	0	A Alk	A	0 or Avs	0 or Avs	0 or Avs	A	A	—	—
<i>B. meladysen- tericus</i> Cas- tellani 1904 var C	0	0	0	0	A Alk D	As	0 or Avs	0 or Avs	0 or Avs	A	As	—	—
<i>B. meladysen- tericus</i> Cas- tellani 1904 var D	0	0	0	0	A Alk	A	A	A	A	A	A	—	—
<i>B. morgani</i> Castellani and Chalmers 1918	0	0	0	0	0 Alk or As Alk	0	0	0	0	AG	0 or A	0 or A	0 or A
<i>B. neapolitanus</i> Emmerich	0	0	0	0	AC	AG	AG	AG	AG	AG	AG	AG	AG
<i>B. negombensis</i> Castellani 1910	0	0	0	0	0 Alk	0	0	0	0	A	0	0	0
<i>B. oxylocus per- nicosus</i> Wya- sokowitsch	0	0	0	0	AC	AG	AG	AG	AG	AG	—	AG	AG
<i>B. para acitryke</i> Castellani 1914	+	0	0	0	A Alk	0	0	AG	AG	AG	AG	0	0
<i>B. para assati- cus</i> Castel- lani 1916	0	0	0	0	0	0	0	AG	AG	AG	AG	AG	AG
<i>B. paracoagu- lans</i> Castel- lani 1914	0	0	0	0	AC	0	0	0	AG	AG	AG	—	AG
<i>B. paracolon</i> Day	+	0	0	0	A Alk	0	0	A	AG	AG	AG	AG	AG
<i>B. paradiffu- ens</i> Castel- lani	+	0	+	+	Alk D or P	0	AG	0	0 or A	AG	0	0	0

	Galactose	Lactulose	Inositol	Salters	Amygdalin	Isodulcitol	Erythritol	Glycerine	Indol	Yogurt Prosk	Dyala	Remarks
G	AG	AG	O	O	O	—	—	O	O	—	Gt	Identical culturally with <i>B. suspensifer</i> (= <i>B. aertryke</i> ) and <i>B. paratyphosus</i> . <i>B.</i> differs serologically.
O	O	O	O	O	O	O	O	O	O	O	Gt	The typical <i>B. faecalis alkiligenes</i> produces strong alkalinity in all sugar broths but certain strains are said to produce slight acidity in glucose and maltose. Some strains peptonize milk.
A	A	A	—	O	O	—	O	—	O	O	Gt	—
—	—	—	A	—	—	—	—	—	O	+	—	Incompletely described. It is probably very similar to <i>B. colotropicalis</i> but indol O.
O	A	O	O	O	O	O	O	O	O	O	Gt P	—
AG	AG	AG	O	AG	O	AG	O	As	+	O	Gt	—
AG	AG	AG	O	—	—	—	O	—	+	O	Gt	—
AG	AG	AG	—	—	—	—	—	—	±	O	Gt	Considered to be identical or with <i>B. suspensifer</i> but complete serological tests have not been carried out.
O	A	A	A	O	O	A	A	A	O	O	Gt	—
AG	AG	AG	O	AG	O	—	—	AG	+	O	—	—
AG	AG	AG	AG	AG	—	—	—	—	O	+	Gt	Differs from <i>B. acidilactici</i> in fermenting inosite.
AG	AG	AG	O	AG	—	—	O	—	O	+	—	—
O	A	A	O	O	O	O	O	As	+	O	Gt	—
A	A	A	O	O	O	A	O	A	+	O	Gt	—
—	Alk	Alk	—	—	—	—	—	—	±	—	Gt	Differs from <i>B. faecalis alkiligenes</i> in being non motile.
AG	AG	AG	AG	AG	O	AG	O	AG	+	O	Gt	Differs from <i>B. pseudo-coli</i> in fermenting inosite.

Bacteria	Motility	Gram	Gelatine	Serum	Litmus Milk	Lactose	Saccharose	Dulcete	Mannite	Glucose	Maltose	Dextrin	Raffinose
<i>B. paradysericus</i> Castellani 1904	O	O	O	O	A	O	O	O	O	O or A	O	O	O
<i>B. paratyphosus</i> Castellani 1914	+	O	O	O	A	AG	AG	AG	AG	AG	AG	AG <sub>s</sub>	AG
<i>B. paratyphosus</i> Castellani	+	O	O	O	AC	AG	O	O	AG	AG	AG	AG	AG
<i>B. paratyphosus</i> A Schotmuller	+	O	O	O	A	O	O	AG	AG	AG	AG	AG	O
<i>B. paratyphosus</i> B Schotmuller	+	O	O	O	A Alk	O	O	AG	AG	AG	AG	AG	O
<i>B. paratyphosus</i> C	-	-	-	-	-	-	-	-	-	-	-	-	-
<i>B. pneumoniae</i> Friedlaender	O	O	O	O	AC	A	AG	AG	AG	AG	AG	AG	AG
<i>B. prinisi</i> Castellani	+	O	O	O	A	O	O	O	O	A	A	A	O
<i>B. proteus vulgaris</i> Hauser	+	+	+	+	C or P	O	AG	O	O	AG	AG	O	O
<i>B. pseudo-asialticus</i> Castellani 1913	O	O	O	O	A Alk	O	AG	AG <sub>s</sub>	AG	AG	AG	AG	AG
<i>B. pseudo-asialticus mobilis</i> Castellani, 1915	+	O	O	O	A or O Alk	O	AG	AG	AG	AG	AG	AG	AG
<i>B. pseudo-carolinus</i> Castellani 1917	O	O	O	O	O	O	O	O	AG	AG	AG	-	AG
<i>B. pseudo-coli</i> Castellani, 1909	+	O	O	O	AC	AG	AG	AG	AG	AG	AG	AG <sub>s</sub>	AG
<i>B. pseudo-coli formis</i> Castellani, 1917	+	O	O	O	AC	AG	O AG	AG	AG	AG	AG	AG	AG

Sorbito	Galactose	Lrvalose	Inosite	Sulcitro	Amalgalim	Isodulcilo	Erythrite	Glycerine	Indol	Loges Frook	Brotb	Remarks
AG	AG	AG	O	O	O	AG	O	AG	+	-	-	-
-	O or As	O	-	O	-	-	-	-	-	-	-	-
-	As	A	-	-	-	-	-	-	+	-	Gt	-
-	A	A	-	-	-	-	-	-	+	-	-	-
-	A	A	-	-	-	-	-	-	O	-	-	-
-	A	A	-	-	-	-	-	-	+	-	-	-
O	A or A or AGs AGs	O	O	O	O	O	O	O	++	O	Gt	-
AG	AG	AG	O	AGs	O	AGs	O	AGs	+	O	-	Differs from <i>B coli</i> in being non motile and in fermenting saccharose from <i>B pseudo-coli</i> in being non motile from <i>B colitrop colis</i> in fermenting dulcilo and saeccharose
O	A	A	O	O	O	O	O	O	O	O	Gt	-
AG	AG	AG	AG	AG	O	AG	O	AG	+	+	-	-
O	AG	AG	AG	O	O	AG	O	AG	O	O	Gt	-
O	AG	AG	AG	O	O	O	AG	O	O	+	O	Gt Differs from <i>B assaticus</i> in not fermenting saccharose and in fermenting dulcilo
O	A	AG	AG	-	-	-	-	-	+	O	Gt	-
-	AG	AG	AG	-	-	-	-	-	+	O	Gt	-
-	-	AG	AG	-	O	-	-	-	O	O	-	Gt



Bacteria	Motility.	Gram.	Gelatin.	Serum	Litmus Milk	Lactose.	Saccharose.	Dulcote.	Mannite.	Glucose	Maltose.	Dextrin.	Raffinose.
<i>B. pseudo coloides</i> Castellani, 1916	-	-	-	-	-	-	-	O	-	-	-	-	-
<i>B. pseudo-coloides</i> , var <i>B. Castellani</i>	-	-	-	-	-	-	-	O	-	-	-	-	-
<i>B. pseudo columbensis</i> Castellani, 1917	O	O	O	O	O	O	O	AG	AG	AG	AG	AGs	AG
<i>B. pseudo-morganii</i> Castellani	+	O	O	O	O, Alk	O	O	O	O	AG	O	O	O
<i>B. pseudo-uesenbergii</i> Castellani, 1918	O	O	O	O	O	O	AG	O	O	AG	-	-	-
<i>B. psittacosis</i> Nocard	+	O	O	O	A, Alk	O	O	AG	AG	AG	AG	AG	AG
<i>B. pyogenes fecidus</i> Passet	+	O	O	O	AC	A	A	A	A	A	A	A	A
<i>B. schaefferi</i> von Freudenreich	O	O	O	-	AC	AG	O	AG	-	-	-	-	-
<i>B. suspensifer</i> Kruse	+	O	O	O	A, Alk	O	O	AG	AG	AG	AG	O	O
													AGs
<i>B. talavensis</i> Castellani, 1909	+	O	O	O	Alk, D	O	A	O	O	A	O	O	O
<i>B. tangallensis</i> Castellani, 1911	O	O	O	O	As, Alk	O	A	A	A	A	A	A	A
<i>B. tardus</i> Castellani, 1917	O	O	O	O	DP	O	O	O	O	As	O	-	-
<i>B. typhi mursum</i> Loeffler	+	O	O	O	A, Alk	O	O	AG	AG	AG	AG	AG	O
													AG
<i>B. typhosus</i> Eberth	+	O	O	O	A	O	O	O	A	A	A	As	O
<i>B. veboda</i> Castellani, 1909	+	O	O	O	A, Alk	O	O	AG	A	AG	AG	AG	AG
<i>B. vebanda</i> Castellani	+	O	O	O	A	AG	O	AG	AG	AG	O	O	AG
<i>B. vesiculosus</i> Henrici	O	O	O	O	AC	AG	O	O	-	-	-	-	O

	Sorbitol	Galactose	Lactulose	Inositol	Salicin	Amygdalin	Isodulcitol	Erythritol	Glycerol	Indol	Voges Prosk	Broth	Remarks
	O or As	O	O	O	O	O	O	O	O	±	—	Gt	Milk rendered permanently acid
)	AG	AG	AG	—	—	—	—	—	—	+	O	Gt	<i>B. bradlownii</i> Castellani 1911 culturally identical differs serologically
)	AG	AG	AG	O	AG	O	AG	O	AG	±	—	Gt	Differs from <i>B. grunthii</i> in fermenting maltose
)	AG	AG	AG	O	O	O	AG	O	O or As	O	O	Gt	—
)	AG	AG	AG	AG	O	O	AG	O	O	O	O	Gt	Certain strains serologically typical may produce at times only instead of AG some strains do not ferment inositol (Weiss and Rice)
	—	—	—	—	—	—	—	—	—	—	—	—	Cover several groups of identical with <i>B. enteritidis</i>
)	AG	AG	AG	AG	AG	—	—	O	—	O	O	—	—
A	O	A	A	O	A	O	O	O	O	O	—	Gt	—
)	O	AG	A or AG	O	O	—	—	—	O or As	+	—	Gt	Cultures emit a disagreeable odour. Strains distinguished at first three varieties of proteus <i>P. vulgaris</i> (rapid liquefaction of gelatin) <i>P. mirabilis</i> (slow liquefaction) <i>P. zenkeri</i> (no liquefaction) later abandoned thus differentiation)
O	AG	AG	AG	B	AG	O	AG	O	AG	+	O	Gt	Differs from <i>B. asiatica</i> in fermenting dulcitol
O	AG	AG	AG	O	AG	O	AG	O	AG	+	O	Gt	—
O	A	AG	AG	—	—	—	—	—	—	+	O	Gt P	—
O	AG	AG	AG	O	AG	O	AG	O	AGs	+	O	Gt	Differs from <i>B. coli</i> in fermenting saccharose belonging to the group Communion of coliform bacilli
O	AG	AG	AG	O	AG	O	AG	O	AG	+	O	Gt	Differs from <i>B. pseudo-coli</i> serologically and in fermenting saccharose only after several days

<i>Bacteria</i>	Motility	Gram.	Gelatin	Serum	Litmus Milk	Lactose	Saccharose	Dulcitate	Mannite	Glucose	Maltose	Dextrin	Raffinose
<i>B watayeka</i> Castellani	+	O	O	O	A	O	O	AG	AG	AG	AG	O	AG
<i>B werahensis</i> Castellani	+	O	O	O	A	O	O	AG	A	A	AG	AG	AG
<i>B wesenbergi</i> Castellani	+	—	O	O	A	A	AG	A	A	AG	—	—	—
<i>B wesenbergoides</i> Castellani, 1916	+	O	O	O	O	O	AG	O	O	AG	AG	—	—
<i>B willegodasi</i> Castellani	+	O	O	O	A Alk	O	O	A	A	AG	AG	AG	AG
<i>B wolinskae</i> Castellani 1916	+	O	O	O	A or A Alk	O	A or Alk	O	AG	AG	AG	O	O
<i>B zeylanicus</i> Castellani, 1910	+	O	O	O	Alk	Alk	Alk	Alk	Alk	Alk	Alk	Alk	Alk

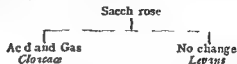
Abbreviations used in the above Table—A=acid, G=gas, C=clot, D=de Peptonized (milk) pellicle (broth), VS=very slight O=negative result—viz, no liquefaction of gelatin or serum as the case may be, + =positive result, ± =±

The new nomenclature has not been used in this table

Sorbito	Galactose	Lactulose	Inosite	Salicin	Amygdalin	Inositol	Erythrite	Glycerine	Indol	Voges Prosk	Broth	Remarks
—	—	—	O	—	—	—	—	—	+	—	—	Differs from <i>B pseudo-coli</i> in not fermenting dulcitate
—	—	—	AG	—	—	—	—	—	+	—	—	
AG	AG	AG	O	AG	O	AG	O	AG	+	O	Gt	—
O	A	A or AG	O	O	O	O	O	O	+	O	Gt	—
—	—	—	—	—	—	—	—	—	+	—	Gt	—
AG	AG	AG	—	O	—	—	—	—	O	O	Gt	Identical with <i>B a rityke</i> according to Bainbridge
—	A	A	—	—	—	—	—	—	+	O	Gt	—
O	—	—	O	—	—	—	—	—	+	O	Gt	Incompletely described
AG	AC	AG	O or AG	O	O	AG	O	As	+s	O	Gt	Identical with <i>B acitryke</i> Other synonyms for <i>B suspect ferare</i> <i>B cholerae</i> <i>sus</i> bacillus of Log cholera Salmon and T Smith 1885
O	A	A	A	A	O	O	O	A	+	O	Gt	—
A	A	A	O	A	O	A	O	A	+	O	Gt	—
—	O	O or As	—	O	—	—	—	—	O	—	—	Very slow and scanty growth on agar
AG	AG	AG	—	O	O	—	—	O	O	O	Gt	Bainbridge has found out that the name is applied to different organisms some strains being serologically identical with <i>B acitryke</i> others with <i>B enteritis</i> <i>dis</i> Gaertner others with <i>B paratyphosus</i> <i>B</i> Aik
A	A	A	O	O	O	O	O	As	O	O	Gt	Certain strains milk A Aik
AG	AG	AG	AG	O	O	A	O	O	O	—	Gt	—
AG	AG	AG	O	O	O	AG	O	AG	O	—	Gt	—
—	—	—	O	—	—	—	—	—	+	O	—	—

Genus *Cloaca* Castellani and Chalmers 1918Type Species — *Cloaca cloacæ* Jordan 1890

Remarks — Two species are known *C. cloacæ* Jordan and *levans* Wolffin but they are not important in tropical medicine. They may be recognized as follows —



## TRIBE BACTERIDIÆ CASTELLANI AND CHALMERS

Definition — Bacillaceæ growing well on ordinary laboratory media without endospores and either fluorescent or chromogenic

Type Genus — *Bacteridium* Schroeter 1872

Remarks — Two distinct groups belong to this tribe—viz the fluorescent and the chromogenic—but we are only concerned with the latter to which the type genus belongs

Genus *Bacteridium* Schroeter 1872

clotted

## TRIBE GRACILOIDÆ CASTELLANI AND CHALMERS 1918

Definition — Bacillaceæ growing very slowly and scantily on ordinary and blood media without endospores or capsules neither fluorescent nor chromogenic

Type Genus — *Graciloides* CastellaniGenus *Graciloides* Castellani 1917Definition — *Graciloides* with the tribal charactersType Species — *Graciloides albofaciens* Castellani 1904

Classification — Two species have been so far described which may be recognized as follows —

A. Litm is mlk rendered acid and clotted — *Albofaciens*

INTESTINAL BACILLI—Continued

Inulin	Sorbitol	Galactose	L inose	Inositol	Salsicin	Amygdalin	Isodulcitol	Erythritol	Glycerine	Indol	Gas Prosk	Broth	Remarks
O	AG	AG	AG	AG	O	O	AG	O	A	+	—	Gt	—
—	—	O	O	—	A	O	AG	—	O	+s	—	Gt	—
—	—	—	—	—	—	—	—	—	—	+	—	Gt	—
—	—	—	—	—	—	—	—	—	—	+	—	Gt	—
—	—	AG	A	—	AG	O	AG	—	O	+s	—	Gt	—
O	O	A or AG	O	O	O	—	—	—	\	O	—	Gt	—
Alk	O or Alk	Alk	Alk	O or Alk	O or Alk	O or Alk	O or Alk	O or Alk	O or Alk	O	O	Gt or P	Classification of the germ being morphologic vibrio bacillus like thrix like spirillum like hence its generic names vibrio spirillum vibrion spirillum vibrion spirillum

Alk=Alkaline S=slight A Alk Acid then alkaline Gt=general turbidity  
 nor clot in milk neither acid nor gas in sugar media non production of indole  
 positive sometimes negative

1. <i>Lactarioides</i> .	Motility.	<i>Listinus</i> Mith.	Lactose.	Saccharose.	Glucose.	Starch.	Indol.	Gelatine.	Egg Albumen.	Broth.	Gram.	Spores.	Appearance of Growth in Deep Glucose Agar.	Formation of Gas in Agar.	Remarks.
<i>B. brunus</i> Distaso	○	○	○	○	○	○	○	○	○	+	+	○	Of a hairy appearance	○	Takes black tint with iodine in the vegetative forms.
<i>B. variabilis</i> Distaso	○	○	S+	S	S+	○	+	○	○	I	○	○	Round and transparent;	+	
<i>B. pseudo ramosus</i> Distaso	○	○	S+	S+	S+	○	+	○	○	I	+	○	Round; non-transparent;	○	
<i>B. anaerobicus</i> Distaso	+	○	○	○	S	○	○	○	○	I	+	○	Almost invisible.	○	
<i>B. cornutus</i> Distaso	+	○	○	○	S+	○	○	○	○	I	○+	○	Almost invisible	○	
<i>B. bulbosus</i> Distaso	+	○	○	○	S+	○	○	○	○	I	○	○	Minute colonies, pin head sized.	○	
<i>B. scitatoriacrom</i> Distaso	+	○	+	○	S	○	+	○	○	I	○	○	Transparent, medium sized	S+	
<i>B. variegatus</i> Distaso	+	○	S+	○	S	○	○	○	○	I	+	○	Minute colonies, pin prick sized	○	
<i>B. difidus</i> Tissier	○	○	+	+	+	○	○	○	○	I	+	○	Picomorphic colonies, mainly round or crenated	○	

Abbreviations used in the Table.—○=negative result; + = positive result; C=clotted; T=turbidity; S+ = slight positive.

## TRIBE BACTEROIDIÆ CASTELLANI AND CHALMERS

**Definition**—Bacillaceæ with good growth on ordinary laboratory media without endospores fluorescence or pigment formation and obligatory anaerobes

**Type Genus**—*Bacteroides* Castellani and Chalmers 1918

**Genus** *Bacteroides* Castellani and Chalmers 1918

## TRIBE BACILLIÆ CASTELLANI AND CHALMERS 1918

**Definition**—Bacillaceæ growing well on ordinary laboratory media and possessing endospores

**Type Genus**—*Bacillus* Cohn 1872 *pro parte*

**Genus** *Bacillus* Cohn 1872

**Definition**—Bacillæ with the tribal characters

**Type Species**—*Bacillus subtilis* (Ehrenberg 1833)

**Classification**—The genus may be divided into two groups as follows—

A Aerobes—*Subtilis* group

B Obligatory anaerobes—*Tetanus* group

Only the latter concerns us at present

## TETANUS GROUP

The group may be divided into subgroups as follows—

A Gelatine liquefied—

I Inspissated blood serum

(a)

(b)

II Inspissated blood serum liquefied—

White of egg digested—*Subgroup Præsolilis*

B Gelatine not liquefied—

Inspissated blood serum not liquefied White of egg not digested  
—*Subgroup Non liquefactæ*

## SUBGROUP QUARTER VIII

A Long threads present—*B. anthracis septimus*

B Long threads absent—

I Saccharose fermented—*B. cereus*

II Saccharose not fermented—

(a) Spores rare in animals—*Novus*

(b) Oval end spores present—*Endosporing types*



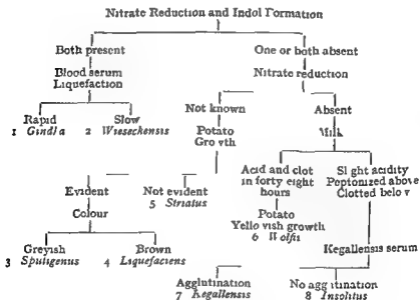
## Series III — A Cholera immune serum

- 1 Agglutination positive
  - 2 Castellani's saturation test equal or
  - 3 Pfeiffer's reaction negative
  - 4 Complement fixation positive
- B Haemolysis feeble and late

The bacteriological characters of the usual vibrio are to be found in every textbook on bacteriology

## GINDHA GROUP

This group may be divided into species as follows —



The names of the eight species so differentiated are —

- 1 *V. gindha* (Pfeiffer 1896)
- 2 *V. liquefaciens* (Migula 1900)
- 3 *V. wieseckensis* (Migula 1900)
- 4 *V. striatus* (Migula 1900)
- 5 *V. wolfi* (Migula 1900)
- 6 *V. spuitigenus* (Migula 1900)
- 7 *V. kegallensis* vel *paracholerae* (Castellani 1913)
- 8 *V. insolitus* (Castellani 1913)

Of these, the first and the last two have been associated with cholera, to the exclusion of other organisms

## SUBGROUP SACCHAROLYTIC

ng stormy  
icken meat

1 *B. welchii* Migula 1900 (Synonyms — *B. perfringens* Veillon and

## A Non motile —

Saccharose and lactose fermented but salicin not fermented—  
*Welchii*

## B Easily motile in cultures more motile in tissues —

I Saccharose and salicin fermented but lactose not fermented—  
*Fallax*

II Saccharose not fermented but lactose and salicin fermented—  
*Aerofetidus*

III Saccharose, lactose and salicin not fermented—*Edematis*

## SUBGROUP PROTEOLYTIC

Definition.—Tetanus group liquefying gelatine and inspissated blood serum Meat media blackened Milk usually digested without forming a clot Colonies grow out in long tangled filaments

Classification.—The following organisms belong to this subgroup —

1 *B. letans* Flügge 1886

2 *B. sporogenes* Metchnikoff 1908 (Synonyms — *B. cadaveris sporogenes* Klein 1901 *B. edematis maligni* Koch 1881 *B. enteritidis sporogenes* Klein 1895 *pro parte B. putrificus coli* Birstock 1906)

3 *B. botulinus* van Ermengem 1898

4 *B. histolyticus* Weinberg and Sequin 1916

They may be divided as follows —

re Pathogenic for

B No formation of white balls in meat media

and feature 17



## FAMILY MYCOBACTERIACEÆ Chester, 1901.

Definition.—Tuberciales with short or long cells, cylindrical or filamentous, without a sheath, but often clavate, cuneate, or

*Mycobacterium* becomes the only genus of the family, as Chester included with it the *Corynebacterium* of Lehmann and Neumann

Genus *Mycobacterium* Lehmann and Neumann, 1896

Definition.—Mycobacteriaceæ with the characters of the family

Type Species.—*Mycobacterium lepræ* (Hansen, 1874)

Remarks.—From our point of view there are two divisions of the genus which are of importance—viz—

A Acid fast when stained by Ziehl Neelsen's method

B Not acid-fast when stained by Ziehl Neelsen's method

while the former are but few in a cell and relatively hard to be colourized. Moreover, the leprosy bacillus can be readily stained by

## REFERENCES.

ANAEROBIC COMMITTEE (1915) Demonstration of Anaerobes. *Lancet*  
 ARCHIBALD (1915) *Lancet* (Weisenberg and Alcaligenes)

new classification in his paper, 'Les Bases actuelles de la Systematique e Mycologie'

With this brief history we will pass on to consider the *Fungaceæ* Linnæus, 1737, which, as we have already seen belongs to the Regnum Vegetabile, division Thallophyta

#### SUBDIVISION FUNGACEÆ Linnæus, 1737

free hypha or are formed by sexual cells which may be enclosed in a fruit or perithecium, formed by the interlacing of mycelial threads without chromatophores or chlorophyl

**Morphology.**—Fungi are Thallophytes without chlorophyl, and do not contain starch or chromatophores. Their vegetative body, or thallus, consists generally of a mass of filaments or threads termed the 'mycelium'. The threads or filaments forming the mycelium are called 'hyphæ'. The mycelial threads or hyphæ may be *septate* or *non-septate*. Their walls do not consist of ordinary

make use of the carbon dioxide of the air and therefore derive their carbonaceous food material from complex organic compounds as

They  
Fungi

he great

## CHAPTER XXXVII

# FUNGACEÆ—PHYCOMYCETES

Preliminary—Ustilaginaceæ—Phycomycetes—Zygomycetes—Mucorales—References

### PRELIMINARY.

THE study of fungi, or *mycology*, as it is often called, includes macroscopic and microscopic forms. The microfungi are those which principally cause disease, which, for this reason, is termed a *mycosis*. Thus 'otomycosis' means a mycosis of the ear, and 'mucormycosis' a disease caused by a *mucor* which is a fungus known to Malpighi in 1686.

The study of the microfungi began in the days of Charles II. when Hooke in 1677 made a lens with which he examined the blighted or yellow specks on the leaves of the damask rose and made excellent drawings of the microfungi which he saw. His book contains a chapter devoted to the 'Blue Mold and the First Principles of Vegetation arising from Putrefaction.'

Malpighi in 1686 has a chapter devoted to 'Plantis quæ in aliis vegetant.'

in France

This slow but sure progress now became very rapid and the only way in which we can trace its evolution is by mentioning the great systematic works which have appeared.

## PLATE V

### CULTURES OF SOME TROPICAL FUNGI

- 1 *ENDODERMOPHYTON CONCENTRICUM* Blanchard 1901 *emendavit*  
Castellani 1911  
Typical culture on glucose agar three weeks old
- 2 *ENDODERMOPHYTON TROPICALE* Castellani 1914  
Old culture on glucose agar
- 3 *ENDODERMOPHYTON INDICUM* Castellani 1911  
Fairly old culture on glucose agar
- 4 *ENDODERMOPHYTON INDICUM* Castellani 1911  
Young culture on glucose agar
- 5 *EPIDERMOPHYTON RUBRUM* Castellani 1909  
Culture on glucose agar
- 6 *TRICHOPHYTON VIOLACEUM* Bodin 1902 VAR *DECALVANS*  
Castellani 1911  
Culture on Sabouraud's agar
- 7 *CLADOSPORIUM MANSONI* Castellani 1903  
Culture on Sabouraud's agar
- 8 *NIGROCOCCUS NIGRESCENS* Castellani 1910  
Culture on Sabouraud's agar

which they call 'actinomycetine'. They state that the injection of this substance produces the same lesions as the fungus. Cenci, Besta, Otto and others have obtained various toxins from fungi.

#### *Monilia balcanica* Castellani

Philo Bloch Truffi and others have prepared trichoplaxton vaccines by killing with heat, and triturating cultures of these fungi. By injecting these vaccines into patients suffering from

affected with sporotrichosis

Widal and Abram have introduced a general diagnostic method

are present in large amount

Other biological reactions—complement fixation etc.—have been described

**Reproduction**—The seeds of the *Phanerozonia* may be said to be represented in the fungi by the roundish or oval shaped bodies called 'spores'. The spores multiply by budding producing daughter spores identical with the parent spores. Under certain conditions

distinguished

1 *Conidia* or *Exospores*—These are non sexual spores which take origin by a process of budding or septation from the extremity of a germinal mycelial hypha or *sporophore*. The spores may all be of the same size or, at other times, some are much larger *macroconidia* others smaller *microconidia*. The conidia are at first always unicellular but later they may divide and become multicellular.

2 *Chlamydoconidia* or *Chlamydoconidia*—These are globose or

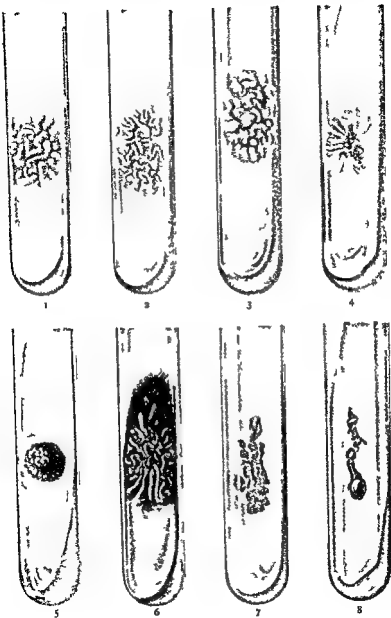
special spore-case structure or *sporangium* which is often terminal and asexual. Endospores which are free and provided with organs of locomotion (cilia or flagella) are called *zoospores* and the sporangium is known under the name of *zoosporangium*.



## PLATE V

### CULTURES OF SOME TROPICAL FUNGI

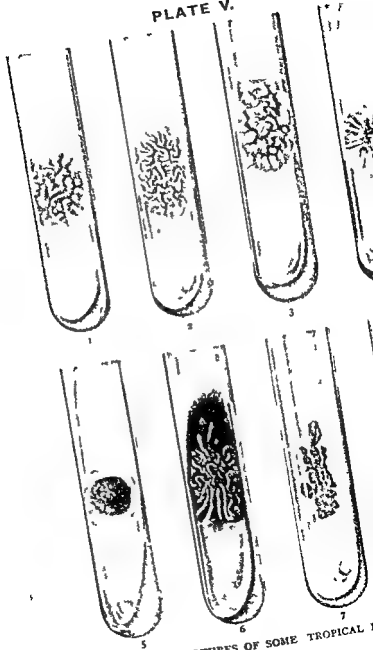
- 1 *ENDODERMOPHYTON CONCENTRICUM* Blanchard 1901 *emendavit*  
Castellani 1911  
Typical culture on glucose agar three weeks old
- 2 *ENDODERMOPHYTON TROPICALE* Castellani 1914  
Old culture on glucose agar
- 3 *ENDODERMOPHYTON INDICUM* Castellani 1911  
Fairly old culture on glucose agar
- 4 *ENDODERMOPHYTON INDICUM* Castellani 1911  
Young culture on glucose agar
- 5 *EPIDERMOPHYTON RUBRUM* Castellani 1909  
Culture on glucose agar
- 6 *TRICHOPHYTON VIOLACEUM* Bodin 1902 VAR *DECALVANS*  
Castellani 1911  
Culture on Sabouraud's agar
- 7 *CLADOSPORIUM MANSONI* Castellani 1905  
Culture on Sabouraud's agar
- 8 *NIGROCOCCUS NIGRESCENS* Castellani 1910  
Culture on Sabouraud's agar



CULTURES OF SOME TROPICAL FUNGI



PLATE V.



CULTURES OF SOME TROPICAL F

## CLASS I PHYCOMYCETES Dr BARY, 1856

**Definition.**—Fungicetes with mycelium continuous in the vegetative stage

**Type Genus** — *Mucor* Micheli, 1729

**Classification.**—The Phycomycetes may be divided into subclasses as follows —

A Sexual spores when present isogamous (similar gametes)—Subclass 1  
*Zygomycetes*

B Sexual spores when present heterogamous (dissimilar gametes)—  
Subclass 2 *Oomycetes*

## ZYGOMYCETES

**Definition.**—Phycomycetes with similar gametes

**Classification.**—The Zygomycetes may be divided into two orders —

A Several asexual spores in sporangia—Order 1 *Mucorales*

B Solitary asexual spore & true conidium on conidiophore—Order 2  
*Entomophthorales*

Only the first order is of interest to us

## ORDER MUCORALES

**Definition**—Zygomycetes with several asexual spores in a sporangium which in some genera are conidia like bodies

**Type Genus** — *Mucor* Micheli 1729

**Classification**—The Mucorales may be divided into the following families —

A Asexual spores in typical sporangia in some genera few spored  
I Columella present zygosporangia naked and thinly covered—Family  
1 *Mucoraceæ*

II Columella absent zygosporangia closely covered by hyphæ—  
Family 2 *Mortierellaceæ*

B Asexual spores not in typical sporangia—Families *Chaenophoraceæ*  
*Chaetocladiaceæ* *Piptocladiaceæ*

Only the Mucoraceæ are of importance to us

## FAMILY MUCORACEÆ

These organisms

called gonidiophores take origin each of which supports on its distal extremity a pear shaped, globular, or claviform sporangium called *gonidangium*. The *sporangium* is at first separated from the gonidiophore by a septum which later protrudes into the lower portion of the sporangium to form a variously shaped structure termed the *columella*.

Inside the sporangium or gonidangium *endospores* or *gonidia* develop by free cell formation

*Ascus Fructifications* or *Asci* are special sporangia containing four or eight or a multiple of eight spores arranged in a single line. These spores are called *ascospores*. Each ascospore presents two membranes *one internal, one external*. The external membrane

give rise to small club shaped branches which come into contact and fuse together forming a new large cell which presents a very resistant double wall. The special branches (sterigmata) which come into contact and unite to produce the spores do not show any apparent sexual differentiation.

5 *Oöspores*—These are formed by a special sexual act of fertilization and can therefore be considered as fertilized ovum.

The female element (*oösporangium*) is usually more roundish protoplasmatic mass (spherical *female gametes*) and presents a thick wall. The male gamete (*antheridium*) which is a special hypha comes into contact with the female protoplasmatic process through it. In contact it divides into several motile bodies called *zoospores* or *zoosporangium* formation of particles.

**Classification**—The *Fungaceæ* may be divided into divisions viz—

- A Vegetative body a multinucleate naked plasmodium—*Myxomycetes*
- B Vegetative body usually filamentous—*Leptoglyphales* Schroeter 1892

We are only concerned with the *Lumycetes*

### **Eumycetes** Schroeter 1892

The *Lumycetes* may be classified as follows

- A Mycelium continuous in the vegetative stage—Class I *Phycomycetes* De Bary 1856
- B Mycelium septate—
  - I Spores in asc—Class II *Ascomycetes* Berkeley
  - II Spores in basidia—Class III *Basidiomycetes* De Bary 1856
  - III Spores not in asc or basidia but on conical spores naked or in pyrenoida or unknown—Class IV *Imperfecti* Focke 1869

The fungi parasitic in man are practically all found among the *Phycomycetes*, the *Ascomycetes* and the *Fungi Imperfecti*. Only one species of importance is found among the *Basidiomycetes* and none among the *Myxomycetes*.

**Mucor pusillus Lindt 1886**

Mycelium at first white then yellowish. The hyphæ carrying sporangia are much shorter than in the preceding species. The sporangium is globular, at first pale greyish then dark greyish. Its diameter varies between 50 and 80  $\mu$ . The columella is claviform, ovoid or spherical, yellowish or brownish. The spores are smooth, spherical, 3 to 3.5  $\mu$  in diameter.



FIG 479—*Mucor mucedo*  
LINNÆUS



FIG 480—*Mucor pusillus* LINDT  
(After Lindt)

*M. pusillus* is often found in bread which has been soaked in water. It is very pathogenic to rabbits; it has once been found in man in a case of otomycosis by Jakowski.

**Genus Lichtheimia Vuillemin 1904**

Non ramified mycelium; rhizoids may be present or not; peduncle supporting sporangium terminates in a formation encircling the base of the columella.

**Lichtheimia corymbifera Cohn 1884**

Synonym—*Mucor corymbifer* Cohn 1884

Mycelium at first white then yellowish. The sporangia are pear-

observed several times in man giving rise to a mycosis of the ear (Huckel, Siebenmann, Graham) of the nose (Siebenmann) of the lungs (Podick). A case of generalized infection has been recorded by Paltauf (see p. 977).

**Lichtheimia ramosa Lindt 1886**

Synonyms—*Mucor ramosus* Lindt 1886; *Lichtheimia ramosa* Vuillemin 1904.

rhizoids are often present  
length. The columella

by Jakowski in a case of  
repeatedly found in the

The sporangial protoplasm not used in the formation of endospores gives rise to a peculiar mucilaginous substance which at a later period by absorption of water causes the bursting of the sporangium. Each endospore or gonidium when it has become

The Mucoraceæ are often the cause of mycosis of plants and animals. A mycosis due to these parasites is often termed mucormycosis.

Mucoraceæ can be easily grown on sugar culture media—for instance Sabouraud's maltose agar—or even on ordinary agar. The optimum temperature for their growth is between 35° and 40° C. The Mucoraceæ require plenty of oxygen and therefore the media tubes must never be closed with rubber caps. When there is not enough oxygen the Mucoraceæ lose their characteristics and give rise to monilia like or yeast like forms.

**Classification**—Four genera of Mucoraceæ are found to contain species parasitic on man—

Family }  
Mucoraceæ

uncle  
rma

ke n

### Genus *Mucor* Micheli 1729

Ramified mycelium absence of rhizoids

#### *Mucor mucedo* Linnæus 1-64

**Synonyms**—*Mucor vulgaris* Micheli 1729 *M. sphaerocephalus* Bulliard 1791

The hyphae carrying sporangia (sporangio-phores) are long and erect the sporangium is globular 100 to 200  $\mu$  in diameter its colour brownish its surface covered by fine minute crystals of oxalate of calcium. The spores (gonidia) are elliptical with a smooth surface. The columella is ovoid shaped and generally yellowish. Occasionally very large zygospores may be observed.

*M. mucedo* L. is very common living in organic substances in



forming a snow white mass Sporangia globular, of black color when ripe Spores ovoid, smooth Columella is at first globular but later takes a cylindrical shape and when the spores have become detached, shows a peculiar mushroom like appearance



FIG 483—*Rhizomucor septatus* VON BEZOLD  
(After Siebenmann)



FIG 484—*Rhizopus niger*  
CIAGLINSKI AND HEWELKE  
(After Ciaglinski)

This species was discovered by Ciaglinski and Hewelke in a case of black mycosis of the tongue

#### General Remarks on Mycoses due to Species of the Family Mucoraceæ.

These mycoses are generally called mucormycoses. They have

cases are on record in  
*Lichtheimia corymbifera*  
nose Their pathogenic

due to *Rhizopus niger*

MUCORMYCOSIS OF THE EAR—OTOMUCORMYCOSIS—Several cases are found in the literature (Siebenmann Boke Hüchel, etc) Almost always *Lichtheimia corymbifera* was present In the tropics we have observed two cases in which *L. ramosa* occurred When the fungus is in great quantity, the patient complains of tinnitus aurium and deafness—the same symptoms as those produced by a plug of cerumen in the external auditory meatus

MUCORMYCOSIS OF THE LUNGS—This condition is rare Furbringer has described two cases in which *L. corymbifera* was

Lucet,  
woman  
rather  
threads  
and the

Genus *Rhizomucor* Lucet and Costantin 1900

Rhizoids generally present columella of ovoid shape

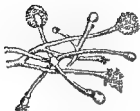
*Rhizomucor parasiticus* Lucet and Costantin 1900

FIG 481 — *Lichtheimia corymbifera* VUILLEMIN  
(After Lichtheim)

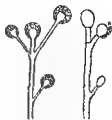


FIG 482 — *Rhizomucor parasiticus* LUCET AND COSTANTIN  
(After Lucet and Costantin)

The columella is ovoid or pyriform dark brownish spores ovoid longitudinal diameter  $4\ \mu$  transverse diameter  $2.5\ \mu$

*R. parasiticus* is pathogenic for guinea pigs and rabbits. It was found in the expectoration of a woman by Lucet Costantin and Lambry. The patient had been considered at first to be suffering from tuberculosis. She recovered under a potassium iodide and arsenical treatment.

*Rhizomucor septatus* von Bezold 1889

Synonyms — *Mucor septatus* von Bezold. *Rhizomucor septatus* Lucet and Costantin 1889

Rhizoids present sporangia of a brownish greyish colour spherical with a smooth or occasionally slightly moriform surface, diameter about 30 to 35  $\mu$  columella spherical brownish spores roundish or slightly oval from 2.5 to 4  $\mu$

The spores are of a yellowish or brownish colour spherical or ovoid with a smooth surface.

This species was found by Siebenmann in a case of otomycosis.

Genus *Rhizopus* Ehrenberg 1820

Rhizoids present columella hemispheric mushroom like

This genus contains only one species

*Rhizopus niger* Criginskii and Hewelke 1893

Synonym — *Mucor niger* Criginskii and Hewelke 1893

The mycelial filaments are provided with abundant rhizoids

## CHAPTER XXXVIII

# ASCOMYCETES AND BASIDIOMYCETES

Preliminary — Classification — Protoascomycetes — Saccharomycetales — Saccharomycetaceæ — Endomycetaceæ — Euscomycetes — Gymnoascaceæ — Aspergillaceæ — Pyrenomyces — Basidiomycetes — References

### PRELIMINARY

THIS chapter includes an account of the fungi parasitic on man which belong to the Ascomycetes. These fungi are characterized by their mode of reproduction—viz by spores originating inside special cells called *asci*. The spores (ascospores endospores gonio- or eight or a multiple of life no *asci* are found and ation and conidia. The fungi belonging to this order are often pleomorphic their morphological characters changing according to the medium on which they live.

**Classification** — The Ascomycetes are divided into subclasses as follows —

- A *Asci* with varying number of spores usually numerous—*Hemiascomycetes*
- B *Asci* with a definite number of spores —
  - I *Asci* separate or scattered—*Protoascomycetes*
  - II *Asci* approximate usually forming a hymenium—*Euscomycetes*

### SUBCLASS PROTOASCOMYCETES

This subclass contains a single order the Saccharomycetales

#### ORDER SACCHAROMYCETALES

This order is divided into two families as follows —

- A Vegetative cells single or loosely attached in irregular colonies—*Saccharomycetaceæ*
- B Vegetative cells forming a mycelium—*Endomycetaceæ*

#### FAMILY SACCHAROMYCETACEÆ REES 1870

**Definition** — Protoascomycetes with vegetative cells single or loosely attached in irregular colonies mycelium not usually

fungus grown. The condition lasted several months. Potassium iodide was given and later, owing to the symptoms of iodism, various arsenical preparations. Castellani has recorded a case of bronchomucormycosis due to *Mucor mucalo* in the Malcanic Zone.

## REFERENCES

## Current Literature

This is very scattered but references can usually be found in the *Bulletin de l'Institut Pasteur* while original papers may be found in the *Archives de*

Revue (1913) P + A D A —

Not and other journals

## Important Old Publications

In preparing this chapter we have used the following —

PERSOON D. C. H. (1801) *Synopsis Methodica Fungorum* Göttingæ  
 ROBIN CHARLES (1853) *Végétaux Parasites* Paris

*Mucor*

CASTELLANI (1903-1914) *Ceylon Medical Reports* (Scattered references)  
 Colombo (1917) *Journal of Tropical Medicine and Hygiene* September  
 (Mucormycosis) London (1918) *Annali Medicina Navale* vol. 1  
 fasc. 1-14  
 ZIMMERMAN (1871) *Das Genus Mucor*, Chemnitz (History, Morphology,  
 Classification)

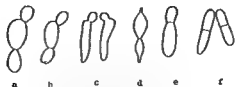


FIG 485—TYPES OF YEASTS

- *Saccharomyces cerevisiae* b, *S. ellipsoideus*, c *S. pastorianus*,  
d ■ *apiculatus* e *Saccharomyces* f *Schizosaccharomyces*.  
(After Lindner)



FIG 486—ZYGOSIS AND FORMATION OF AN ASCUS IN *Zygosaccharomyces octosporus*  
(After Guilliermond)



FIG 487—REPRODUCTION OF SACCHAROMYCES



FIG 488—TYPES OF ASCOSPORES  
1, *Saccharomyces* 2, 4 *Willha*, 5 *Debaryomyces* 6 *Schionniomyces* 7 *Saccharomycesopsis*  
8 *Monospora* ■ *Nematospora*  
(After Guilliermond)

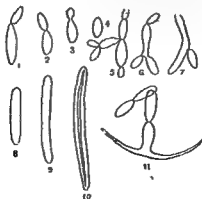


FIG 489—*Monospora cuspidata*

1, 2 Budding cells 8-9 Ascus formation 10, Ascospore germinating

up to 12 spores (ascospores)

on the hyphæ

**Classification**—The family contains thirteen genera, of which only two—*Saccharomyces* Meyen, 1837, and *Willia* Hansen, 1904—contain species parasitic on man, while in regard to *Schizosaccharomyces* Beyerinck, 1893 it appears doubtful to us whether this really should be placed in this family, as it reproduces by division instead of by budding. The various genera may be recognized as follows—

A<sup>1</sup> Vegetative cells globose ovoid or pyriform without lemon-shaped extremities—

B<sup>1</sup>

C<sup>1</sup>

outer exosporium—*Saccharomycopsis* Schüdnning

Γ<sup>2</sup> Ascus formation preceded by zygosis—

Ascospores have a smooth membrane—*Zygosaccharomyces*  
Barker 1901

Π<sup>1</sup> C<sup>2</sup>—

C<sup>2</sup>

B<sup>2</sup>

A<sup>2</sup> Vegetative cells oval with one or both extremities lemon-shaped

A<sup>3</sup>

isolated once by Vuillemin which has all the characteristics of the genus *Endomyces*. Landrien has suggested the term *Endomyces vuillemini*.

### *Endomyces vuillemini* Landrien 1912

**Synonym** — *Endomyces albicans* Vuillemin 1898

Found by Vuillemin in 1898 in thrush patches

**Parasitic Life** — This fungus forms white patches on the tongue and buccal mucosa. The patches are easily detached. A patch examined under the microscope shows septate mycelial threads, simple or ramified, the articles of which are straight or

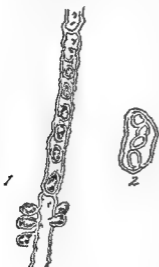


FIG 493 — *Endomyces vuillemini* LANDRIEN

(From a culture. After Vuillemin.)

1 Mycelial threads with chlamydospores and conidia 2 Chlamydospore and conidium

what bent. Each article or cell is about 20  $\mu$  in length and 3 to 4  $\mu$  in breadth. At the terminal portion of a mycelial thread three or four small, ovoid elements are found which reproduce by budding. Some similar to those of *Endomyces* may be seen.

globular elements which were at first considered to be spores, but are now considered to be modified mycelial articles and reproduce by germination.

**Saprophytic Life** — **Culture Characters** — The fungus grows well on slightly acid Sabouraud's and other media. It does not grow abundantly in alkaline media. It does not ferment lactose and is Gram positive.

In cultures the fungus appears in two forms: (1) A filamentous form showing the mycelial threads simple or ramified; (2) A globular form morphologically similar to a typical yeast, reproducing by budding. Both forms may be found in the same culture.

The fungus in cultures reproduces by —

1 Chlamydospores or external spores — large globular cells 15 to 20  $\mu$  in diameter, with thick resistant walls. The chlamydospores are situated at the terminal extremity of some mycelial hyphae and represent modified mycelial articles.

Genus *Saccharomyces* Meyen, 1838

**Definition.**—No proper thallus. Reproduction by budding and ascospores, fermentation of glucose and saccharose, and often of other carbohydrates. Ascospores with one membrane. No trace of any copulative process. Occasionally rudimentary mycelial tubes present, with transverse septation.

**Remarks.**—As already stated, the fungi of this genus, as well as of the genera *Cryptococcus*, *Monilia*, *Oidium* and *Coccidioides* are usually called Blastomycetes, and the diseases induced by them blastomycoses.

The fungi of the genus *Saccharomyces* are of great importance and

of blastomycetic origin

Fungi of the genus *Saccharomyces* are very important from an industrial and agricultural standpoint, being the cause of alcoholic fermentation. The best known yeast is the brewer's yeast *S. cerevisiae* which is slightly ovoid, 8 to 9  $\mu$  in diameter.

*Saccharomyces blanchardi* Guart, 1906

Found at an operation by Blanchard, Schwartz, and J. Binot, on a patient who had been considered to be suffering from tubercular peritonitis. The fungus had produced in the peritoneum

mucoid growth, whitish, darkening after a long time. On carrot growth viscid, abundant, pathogenic to rabbits, in which it induced a general mycosis, terminating fatally.

*Saccharomyces granulatus* Vuillemin and Legrain, 1900

Observed by Vuillemin and Legrain in a tumour of the submaxillary bone. Cells ovoid, 4 to 5  $\mu$  in length, and 3 to 4  $\mu$  in breadth. Cultures pinkish or pinkish red, ascospores and chlamydospores present. Gelatine not liquefied. Sugar reactions not given.

*Saccharomyces tumefaciens* Curtis, 1896

**Synonym.**—*Saccharomyces subcutaneous tumefaciens* Curtis, 1896

Found by Curtis in a myxomatous tumour. It appeared in the tissues in the shape of spherical bodies 16 to 20  $\mu$  in diameter, each surrounded by a zone of amorphous substance. Grows well on all sugar media, in very old cultures asci are seen, with 1-4 spores.



## ORDER ASPERGILLALES

**Definition**—Fungi with asci gathered into definite cylindrical or globose perithecia

**Type Family**—Aspergillaceæ

**Classification**—The order contains a number of families which may be recognized as follows—

A Peridium of loose floccose hyphae—*Gymnoascaceæ* Baranetzky 1872

B Peridium compact—

I Perithecia small—

(a) Perithecia mostly sessile—

1 Peridia closed—*Aspergillaceæ*

2 Peridia open—*Trichocomaceæ*

(b) Perithecia mostly stalked—*Oxygenaceæ*

II Perithecia large—*Claphomyetaceæ* and other families

Only the *Gymnoascaceæ* and the *Aspergillaceæ* concern us

## FAMILY GYMNOASCACEÆ BARANETZKY 1872

**Definition**—Aspergillales with the peridium composed of loose hyphae at the extremities of which the asci are situated or without perithecial or ascial formation and reproducing (as far as known) as a rule by mycelial or conidial spores

It follows—

above it is obvious into two tribes as

A *Ascomycetes* type—

With perithecia and asci—Tribe 1 *Gymnoasceæ* Castellani and Chalmers 1918

B *Fungi Imperfecti* type—

Without perithecia or asci. Reproduction asexual by mycelial and conidial spores—Tribe 2 *Trichophytoneæ* Castellani and Chalmers 1918

The first tribe includes the genera *Mixotrichum* Kunze 1823  
*Gymnoascus* Baranetzky 1872 *Ctenomyces* Eidam 1880 *Amauro*

**Genus *Coccidioides* Rixford and Gilchrist, 1896**

**Definition**—Endomycetaceæ with mycelium well developed and asci containing a large number of spores with intermediate characters between *Saccharomyces* and *Monilia*. One species only *Coccidioides immitis* Rixford and Gilchrist 1896

***Coccidioides immitis* Rixford and Gilchrist 1896**

**Synonyms**—*Coccidioides pyogenes* Rixford and Gilchrist 1896, *Oidium coccidioides* Ophuls 1905, *O. protooides* Ophuls 1905, *Posadasia esferiformis* Canton 1898, *Oidium immitis* Verdun 1907

**Definition**—*Coccidioides* with a large number of spores in the asci

Discovered by Wernicke in 1882 in America in a patient with patches resembling a tubercule and gummatæ. In the lesions roundish bodies are seen of various sizes 3 to 80  $\mu$  in diameter with a well defined thick membrane. Inside some of the bodies numerous spores may be seen. This genus is not well defined.

**Cultures**—The fungus grows under two types (1) a saccharo- a filamentous type  
d but deepening into  
hen old often become  
many spores may be present. The

It probably lives saprophytic in nature

**SUBCLASS EUASCOMYCETES**

**Definition**—Ascomycetes with asci not separate or scattered

recognized —

A. Asci approximate no definite ascoma but an indefinite hymenium—*Protodiscales*

*Aspergillales*

(b) Penthecrium on a short pedicel—*Laboulbeniales*

Only the *Aspergillales* and the *Pyrenomycetes* interest us. The latter differ from the former by having their asci arranged in a hymenium within the closed ascocarp.

and therefore be

they had found oval

The peridium was

at first loose but hardened later and contained a number of asci situate at

the Aspergillaceæ but at that stage they consisted of the outer wall only the contents having all disappeared They therefore considered that their observation supported the views of Matrachot and Dassonville

We therefore classify the genus *Trichophyton* in the *Gymnoascaceæ* but the very large number of species included in that genus exhibit such different characters that they can easily be arranged in groups which appear to us to be of generic value especially as we know that Malmsten meant only the form we now call *endothrix* to be designated by his name *Trichophyton* because he states — The mould formation appears in the root of the hair and it occurs *only* inside of the hair between its fibres so that the epithelial layer is uninfected besides there is no mould formation to be found among the epidermal cells so that one can say with

below

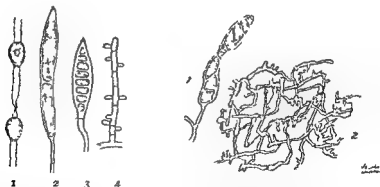


FIG 494 — *Microsporium audouinii* GRUBY (After Bodin)  
 FIG 495 — *Microsporium audouinii* GRUBY (From cultures)

1 Chlamydospores 2 septate spindle 3 pluriseptate spindle 4 spore bearing hypha of *Acladium*  
 1 Septate spindle body 2 mycelium with denticulated structures

#### TRIBE TRICHOPHYTONÆ Castellani and Chalmers 1918

Definition — *Gymnoascaceæ* of the *Fungi Imperfecti* type

Type Genus — *Trichophyton* Malmsten 1845

We however classify them as given above and our reasons for so doing may be set forth in a short historical statement

5      10      15      20      25

these classifications of Schroeter a more detailed arrangement of the Ascomy  
 cetes be adopted does not concern our present purpose as both contain the

form

— 30 — 35 — 40 — 45 — 50 — 55 — 60 — 65 — 70 — 75 — 80 — 85 — 90 — 95 — 100 —

Genus *Microsporium* Gruby, 1843.

**Definition.**—Gymnoascaceæ with only Fungi Imperfecti forms known, producing in the lesions mycelial filaments and mycelial

yellow favic scutula

oval, 3 to 4  $\mu$  in length, and 2 to 3  $\mu$  in breadth. They are not supported by short sterigmata, as is the case in the genus *Trichophyton*.

2. *Terminal Septate or Non Septate Spindles*—These structures are found at the end of certain filaments and may be considered to be modified terminal conidia. They are large fusiform structures, 30 to 60  $\mu$  in length, and 15 to 18  $\mu$  in breadth. They have granular contents, and may be septate or non septate. The surface, especially at the apex, presents some peculiar hair-like formations which are not observed in the spindles of the fungi of the genus *Trichophyton*.

3. *Chlamydospores*—Certain mycelial articles become expanse, ovoid, or ampulliform, 12 to 18  $\mu$  in length, and 6 to 8  $\mu$  in breadth.

Their much chlam tate 1

become ts are vege often

seen in fungi of the genus *Microsporium* are the so called *denticulate* or *pectinate bodies*, which are mycelial segments, generally curved, showing on one side—the convex side as a rule—several small protruding processes. Bodin has demonstrated these processes to be mycelial tubes arrested in their development.

**Pleomorphism.**—After a time, and often rapidly, the cultures of microsporons lose their characteristics and become pleomorphic. This phenomenon is not very prominent, however, in the most important species—viz, *Microsporium audouinii*—while it is very accentuated in some microsporons of animal origin, especially *M. minimum*, in which, according to the complete investigation carried out by Bodin, two types may be met with—

1. THE DOWNY TYPE (*Acladium type*)
2. THE GLABROUS TYPE (*Endoconidium type*)

**Classification.**—The various genera belonging to this tribe may be recognized as follows —

A *In the ...* — not ... — Genus *Lopho*

B *In the ...* not attack hairs of hair follicles but grow in the superficial or deep strata of the epidermis

(a) Pluriseptate spindles present in cultures. Grow in the superficial strata of the epidermis do not attack hairs. Cultures

often pyogenic and of animal origin—  
Genus *Eciotrichophyton* Castellani and  
Chalmers 1918

(ii) G

(\*) Do not attack hairs or hair follicles — Genus

2 It

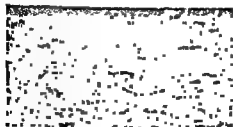


FIG 496—*Trichophyton cerasi* CHALMERS AND MARSHALL, TO SHOW FUNGUS IN HAIR

Genus *Microsporium* Gruby 1843

**Definition**—Gymnoascacea with only 1 ungi Imperfecti forms known producing in the lesions mycelial filaments and mycelial spores which are small roundish and about 2-3 microns in diameter. In cultures hyphae bearing sessile conidia may be seen and septate or non septate fusiform bodies. The lesions in man do not produce yellow favic scutula.

spores being roundish small (2 to 3  $\mu$ ) and irregularly arranged in a mosaic like manner. They are easily cultivated on Sabouraud's and other sugar media (see Genus *Trichophyton* p. 996). In cultures the microsporons proliferate by sprouting with branching of the mycelium and also often by—

1 *Spore Bearing Hyphae*—of type *Acladium* the conidia being oval 3 to 4  $\mu$  in length and 2 to 3  $\mu$  in breadth. They are not supported by short sterigmata as is the case in the genus *Trichophyton*.

2 *Terminal Septate or Non Septate Spindles*—These structures are found at the end of certain filaments and may be considered to be modified terminal conidia. They are large fusiform structures 30 to 60  $\mu$  in length and 15 to 18  $\mu$  in breadth. They have granular contents and may be septate or non septate. The surface especially at the apex presents some peculiar hair like formations which are not observed in the spindles of the fungi of the genus *Trichophyton*.

3 *Chlamydospores*—Certain mycelial articles become expanse ovoid or ampulliform 22 to 18  $\mu$  in length and 6 to 8  $\mu$  in breadth. Their protoplasm may be granular and the membrane may become much thicker and have a double contour. These elements are chlamydospores. They are generally found when the fungi vegetate under unfavourable conditions. Another structure often seen in fungi of the genus *Microsporium* are the so called *denticulate* or *pectinate bodies* which are mycelial segments generally curved showing on one side—the convex side as a rule—several small protruding processes. Bodin has demonstrated these processes to be mycelial tubes arrested in their development.

**Pleomorphism**—After a time and often rapidly the cultures of microsporons lose their characteristics and become pleomorphic. This phenomenon is not very prominent however in the most important species—viz *Microsporium audouinii*—while it is very accentuated in some microsporons of animal origin especially *M. minimum* in which according to the complete investigation carried out by Bodin two types may be met with—

1 THE DOWNY TYPE (*Acladium* type)

2 THE GLABROUS TYPE (*Endocoridium* type)

THE DOWNY TYPE IS FOUND ON THE ORDINARY SUGAR MEDIA IN

(*larium*) DO NOT SEEM TO BE INCORPORABLE INTO THE FLOWER AND FRUIT

TABLE SHOWING SPECIES OF MICROSPORUM PARASITIC ON MAN

Genus		Species
Microsporium Gruby 1843	of human origin	<i>M. audouini</i> Gruby 1844
		<i>M. velveticum</i> Sabouraud 1907
		<i>M. umbonatum</i> Sabouraud 1907
		<i>M. tardum</i> Sabouraud 1909
		<i>M. scorteum</i> Priestley 1914
	of animal origin	<i>M. minimum</i> Le Calvé et Mal herbe 1898
		<i>M. lanosum</i> Sabouraud 1907
		<i>M. felineum</i> C Fox and Blacall 1896
		<i>M. fulvum</i> Urbura 1907
		<i>M. pubescens</i> Sabouraud 1909
		<i>M. villosum</i> Linne 1907
		<i>M. tomentosum</i> Pelagatti 1909
		<i>M. iris</i> Pasi 1912
		<i>M. flavescens</i> Horta 1912
<i>M. depauperatum</i> Guég en 191		

Some species (*M. audouini*, *M. velveticum*, *M. umbonatum*, *M. tardum*) seem to be parasitic on man only others (*M. lanosum*, *M. felineum*, *M. minimum* etc) are parasitic on the lower animals but occasionally infect man

#### *Microsporium audouini* Gruby 1843

This parasite was described by Gruby in 1843 but his investigation was forgotten till Sabouraud in his classical researches on ringworm in 1892 demonstrated the plurality of species of



**Saprophytic Life—Cultures**—*Microsporum audouinii* grows well on Sabouraud's maltose agar and other media. The rate of growing is slow. In maltose agar the growth becomes evident about a week after inoculation under the appearance of a plaque of a so-called 'satiny aspect,' beneath the surface. In a few days more aerial hyphæ develop, extending above the surface. When the development is complete—generally this takes about six to eight weeks—the growth is roundish, covered with short greyish duvet, and presents often a central knob and some concentric rings of a whitish greyish colour. The cultural characters, however, are variable, and pleomorphism occurs. The cultural characters have

the colour of dried blood

and, there being spore-bearing hyphæ of type Acladium (see Fig. 494)

3. By large unilocular or multilocular spindle conidia. These spindles are large structures 30 to 60  $\mu$  in length and 15 to 18  $\mu$  in breadth. They may be septate or non-septate.

for and presents a sheath with an opaque whitish sheath. It seldom attacks glabrous parts of the body. *M. audouinii* is extremely common in England, but rare in the South of Europe (Italy), and extremely rare in the tropics. We have never seen cases of ringworm due to *M. audouinii* in Equatorial Africa or Ceylon, but a few cases have been described in Brazil, in Madagascar, and in Senegal.

*M. audouinii* seems to live only on the human subject, but closely allied species have been found by Fox in cats, by Bodin in dogs, and by Bodin, Fox, and others in horses.

#### *Microsporum velveticum* Sabouraud, 1907

the  
No  
common in North America

#### *Microsporum umbonatum* Sabouraud, 1907

Found by Sabouraud in two cases of microsporiasis of the scalp contracted in Russia. The appearance of the growth when it has reached complete development (about twenty-five to thirty days after inoculation) has been compared by Sabouraud to the appearance of an ancient round shield, with a central conical formation representing the *umbo* of the shield. Not inoculable into guinea-pigs.

1. THE DOWNY TYPE IS FOUND ON THE ORDINARY SUGAR MEDIA IN

2 THE GLABROUS TYPE IS OBSERVED ON WORT AGAR

lower animals  
The human  
*umbonatum*, *M*  
*larium*) DO NOT SEEM TO BE INOCULABLE INTO THE LOWER ANIMALS

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		<i>M. tardum</i> Sabouraud 1909
		<i>M. scorteum</i> Priestley 1914
	of animal origin	<i>M. minimum</i> Le Calvé et Mal herbe 1898
		<i>M. lanosum</i> Sabouraud 1907
		<i>M. felineum</i> C Fox and Blyskal 1896
		<i>M. fulvum</i> Urburu 1907.
		<i>M. pubescens</i> Sabouraud 1909.
		<i>M. villosum</i> Minne 1907
		<i>M. tomentosum</i> Pelagatti 1907
		<i>M. ins</i> Passin 1912
		<i>M. flavescens</i> Horta 1912
<i>M. depauperatum</i> Guéguen 1912		

Some species (*M. audouini*, *M. velveticum*, *M. umbonatum*, *M. tardum*) seem to be parasitic on man only others (*M. lanosum*, *M. felineum*, *M. minimum* etc) are parasitic on the lower animals but occasionally infect man

#### *Microsporium audouini* Gruby, 1843

This parasite was described by Gruby in 1843 but his investigation was forgotten, till Sabouraud in his classical researches on ringworm in 1892, demonstrated the plurality of species of the fungi found in this affection and showed that a form of tinea capitis was due to the microsporon described by Gruby

**Parasite Life.**—Around the affected hairs the fungus forms by means of its mycelial spores a white opaque sheath extending 2 or 2 millimetres above and below the hair.

or some furrows radiating from the centre may be present. The central portion which is covered with rather long white duvet is encircled by a zone of powdery appearance outside this there is the peripheral zone covered by the extremely delicate characteristic silky like duvet.

#### *Microsporum villosum* Minne 1907

Found in Belgium by Minne in a child suffering from ringworm of the hair clinically identical with the type caused by *M. audouinii*. On Sabouraud's agar the growth at complete development is about 6 centimetres in diameter the central portion is flattened with powdery slightly brownish surface. This is surrounded by mammillary downy formations which decrease in size towards the periphery.

#### *Microsporum tomentosum* Pelagatti 1909

Enth Pelagatti 1909

..

may be umbilicated. The whole surface is covered by thin white duvet. The central portion will

#### *Microsporum iris* Pasini 1911

Discovered by Pasini in Italy in some cases of microsporiasis capitis clinically somewhat different from the usual type. The fungus grows well on Sabouraud's agar. At complete development—twenty two to twenty six days from inoculation—the growth presents a central knob covered with white duvet and surrounded by white and brick red rings alternating.

The microsporiasis capitis due to this microsporon is characterized by the hairs remaining nearly of normal length and presenting a white greenish discoloration.

#### *Microsporum flavescens* P. Horta 1912

Isolated by Horta from some circinate squamous patches on the neck of a child in Brazil. Grows rapidly on Sabouraud's agar the growth is of a yellow colour there is a depression at the centre from which radiate four or five shallow furrows. Pleomorphic duvet appears quickly.

#### *Microsporum depauperatum* F. Guéguen 1912

Isolated by Guéguen from some circinate dry squamous patches. In cultures the membrane of some nuclear filaments presents peculiar thickenings. Spore bearing hyphae are not so well differentiated as in other species.

**Microsporium tardum** Sabouraud, 1909

Found  
 identical  
*M. audouini*  
 the duvet being shorter

**Microsporium lanosum** Sabouraud, 1907

tinea corporis

On Sabouraud's agar the growth is at first similar to *M. audouini*, only more abundant and more downy. Later—twenty five to thirty days from inoculation—the central portion of the growth becomes umbilicated, the depression being surrounded by a ring of snow white duvet, which in very old cultures may become yellowish. Pleomorphism is common.

**Microsporium felineum** C. Fox and Blaxall, 1896

Common in the cat in England, North America and Belgium, may infect man, attacking hairy and non hairy regions of the body. On Sabouraud's agar the growth is rather abundant, discoid, with flattened surface, showing no furrows and covered with a large amount of grey duvet. The fungus is easily inoculable into cats, dogs, and guinea pigs.

**Microsporium minimum** Le Calvé and Malherbe 1898

**Synonyms**—*Microsporium audouini* var. *equinum* Bodin 1896,  
*Trichophyton minimum* Le Calvé and Malherbe, 1898

to a mild  
 furrowed,  
 or, when  
 present, is very scanty, very short, and of a pinkish colour

**Microsporium fulvum** Uriburú 1907

Found by Uriburú in cases of tinea capitis in the Argentine. It grows very rapidly on Sabouraud's agar, the growth presenting a central umbilico, or projection surrounded by a brownish powdery ring. The peripheral zone of the growth is covered with white duvet.

**Microsporium pubescens** Sabouraud 1909

Discovered by Sabouraud in a case of tinea capitis contracted in New York. It grows rapidly on Sabouraud's medium, the culture being characteristically abundant but with a delicate silky duvet dries from inoculation—

fragile mycelium To this type belongs for instance *Trichophyton sabouraudi* R Blanchard

When the mycelial spores are square the filament straight and its articles long the mycelium is called resistant This type is

of which is the following —

Maltose	4 grammes
Peptone Chassa ng	2 gramme
Agar	1 50 grammes
D st lled water	100 c c

On this medium however pleomorphism is of frequent occurrence

**Pleomorphism** — Cultures on maltose and other sugar agars of all *Trichophytons* with the single exception of *T sabouraudi* becoming old lose their characteristics and become covered with abundant white duvet In these cultures which can be considered degenerate

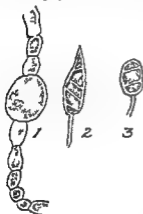


FIG 499 — TRICHOPHYTON



FIG 500 — FRUCTIFICATIONS



FIG 501 — TRICHOPHYTON SPIRAL BODIES

(Preparation from cultures after Bodin)

1 Chlamydospore 2 and 3 septate spindle bodies

1 2 Spore bearing hyphae (*Trichophyton*) 3 spore bearing hypha (*Allopsporium*)

the original type even by animal inoculations

To prevent pleomorphism Sabouraud advises the following medium —

Agar	1 8 grammes
Peptone Chassa ng	3 to 5 grammes
Water	100 c c

**Microsporium scorsteum Priestley, 1914**

" " " " iburu 1909 both morpho-  
 dential therewith as the  
 is parasite are scanty It

*lacticolor* were found Spirals were frequent and pectinate bodies  
 rare Duvet was formed

**Genus Trichophyton Malmsten 1845**

**Definition**—Trichophytonæ with mycelial filaments and spores  
 present in the lesions and conidial bearing hyphæ in cultures only  
 attacking hairs and entirely  
 of human origin Almost  
 never pyogenic

**Type Species**—*Trichophyton*  
*tonsurans* Malmsten 1845

**General Considerations**—  
 During their parasitic life the  
 species of the genus *Tricho*



FIG 497—*Trichophyton curvis*  
 TO SHOW MYCELIAL SPORES



FIG 498—*Trichophyton curvis*  
 TO SHOW CONIDIAL BEARING HYPHA

phyton vegetate according to two types (1) mycelial filaments  
 (2) mycelial spores

The mycelial filaments consist of long cylindrical cells separated  
 by septa The so called mycelial spores are simply a modification  
 of the mycelial filaments due to the septa being much closer so  
 th it the cells limited by them are almost as broad as they are long  
 The term mycelial spores is incorrect as they are not organs of

lating mycelia  
 n appearance  
 type is called

- 1 *T tonsurans* Malmsten 1845
- 2 *T sabouraudii* R Blanchard 1895
- 3 *T violaceum* Bodin 1902
- 4 *T sulphureum* C Fox 1908
- 5 *T glabrum* Sabouraud 1909
- 6 *T sumatum* Sabouraud 1909
- 7 *T effractum* Sabouraud 1909
- 8 *T circumolutum* Sabouraud 1909
- 9 *T regulare* Sabouraud 1909
- 10 *T umbilicatum* Sabouraud 1909
- 11 *T exsiccatum* Uriburu 1909
- 12 *T polygonum* Uriburu 1909
- 13 *T soudanense* Joyeux 1912
- 14 *T curvis* Chalmers and Marshall 1914

These may be recognized as follows —

A

II

(a) *STUDU V SION*

(b)

*l c m ab e a*

II

(a) *STUDU V SION*

*l c m*

(b)

es by  
ou as

On this medium the growth of the various *Trichophytons* is much less abundant than on sugar media but the cultures are fairly characteristic and do not become pleomorphic.

**Experimental Inoculations**—Certain *Trichophytons* can be easily inoculated experimentally into man and many of the laboratory animals—guinea pigs, rabbits etc. Sabouraud advises the inoculation of portions of the cultures to be made into a small flictena artificially induced by burning such as by applying to the skin a lighted match.

The *intravenous* injection may induce generalized lesions of the internal organs.

The intraperitoneal injection as done by Citron may induce a type of peritoneal pseudo-tuberculosis.

**Mode of Infection**—Infection may take place from man to man—this is generally the case with *Trichophytons* of the group *endothrix*—or from the lower animals to man. There is also little doubt that *Trichophytons* may live saprophytically in nature this explaining sporadic cases of trichophytoses in man.

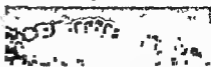


FIG 502—*Trichophyton curvis* TO SHOW LADDER LIKE ROWS OF MYCELIAL SPORES



FIG 503—*Trichophyton curvis* FOUR DAYS GROWTH ON SABOURAUD'S MALTOSÉ AGAR AT 34° C TO SHOW WHITE GROWTH WITH KNOB-LIKE CENTRE



FIG 504—*Trichophyton violaceum* VAR. *tharlotiense*

{Note absence of acuminate centre and dark [violet] colour of the growth }

**Reproduction**—This takes place by—

1 Lateral and terminal conidia supported by short sterigmata

2 Chlamydospores these are rare

3 Large terminal septate and unseptate spindles

**Classification**—The principal species of the genus *Trichophyton* arranged chronologically are —



**Trichophyton soudanense** C Joyeux 1912

Endothrix very similar to *T tonsurans*. In the hairs the mycelial spores are generally rectangular arranged in long strings. On Sabouraud's agar the growth appears three to four days after inoculation as a small yellow nodule later the peripheral portion of the growth appears white. It has been found by C. Joyeux in cases of tinea capitis in the Sudan.

**Trichophyton violaceum** Bodin 1902

Discovered by Sabouraud in cases of tinea barbæ type Endothrix colour

This

a variety of this fungus produces a type of very common ring worm of the scalp in children with white patches covered by enormous numbers of pityriasis squamæ. The patches often remain permanently bald. The Ceylon fungus is endoectothrix and although culturally is hardly distinguishable from *T violaceum* is probably a different variety (var *decalians* Castellani 1913) as it produces lesions generally different from those induced by *T violaceum*. In the Sudan it apparently liquefies gelatine more rapidly (var *khartoumense* Chalmers and Macdonald 1915).

**Trichophyton glabrum** Sabouraud 1909

Closely allied to *T violaceum* but shows a more rapid growth and no violet pigmentation develops. Surface smooth and moist.

**Trichophyton sulphureum** C Fox 1908

Described by Colcott Fox in some cases of tinea capitis in England. Endothrix type. On Sabouraud's agar the growth is characterized by a central reddish nodule which later becomes crateriform assuming a speckled appearance. The rest of the culture has a delicate but distinct primrose or sulphur colour.

**Trichophyton plicatile** Sabouraud 1909

Colonies closely resemble those of *T tonsurans* but have a creased appearance. Found by Sabouraud in cases of sycosis. Sequeira has observed it in a case of trichophytic granuloma. We have placed the fungus in the genus *Neotrichophyton* (p. 1001).

**Trichophyton circinvolutum** Sabouraud 1909

Endothrix somewhat similar to *T plicatile* the growth has convoluted surface. Found by Sabouraud in cases of trichophytosis contracted in the Sudan and Dahomy.

**Trichophyton exsiccatum** Uruburu 1909

Found in Argentina by Uruburu. Endothrix very slow growth crateriform colonies with surface finely cracked and of a dry aspect.

**Trichophyton tonsurans** Malmsten 1845

scalp and the stumps are variously bent. The diseased hairs have a powdery greyish appearance and on pulling them out the roots are not black as in normal hairs. It belongs to the type Endothrix. The mycelial cells are large (4 to 5  $\mu$ ) quadrangular (so called resistant mycelium type). Grows well on maltose agar and other media on maltose agar colonies are white or yellowish often crateriform and present a velvety surface at first later powdery. In hanging drop cultivations spore bearing fructifications can be seen.

*T. tonsurans* besides producing a type of tinea capitis produce also a form of tinea corporis and a trichophytosis of the nails.

**Trichophyton sabouraudi** R Blanchard 1895

Synonym — *Trichophyton acuminatum* Bodin 1902. This is the



FIG 505 — *Trichophyton tonsurans* MALMSTEN

(Preparation of a hair in liquor potassae after Sabouraud)

black dots

**Trichophyton pilosum** Sabouraud 1909

Very similar to *T. sabouraudi* from which it differs only by the cultures being covered when old by a dense short white duvet

the  
over  
mp  
r a



*Trichophyton polygonum* Uriburu 1909

*Endothrix* The growth is at first roundish then takes a characteristic polygonal outline The central part is crateriform

*Trichophyton regulare* Sabouraud 1909

*Endothrix* very similar to *T tonsurans* the cultures being at first crateriform then the edges of the crater becoming undermined the growth takes a peculiar punch like shape with several radiating small sulci The characters of the fungus show always the greatest regularity never changing hence the name *T regulare* given to it by Sabouraud This fungus was found by Dalla Favera

*Trichophyton umbilicatum* Sabouraud 1909

*Endothrix* cultures are deeply umbilicated present at the periphery fine radiating hyphæ forming a sort of aureola

*Trichophyton fumatum* Sabouraud 1909*Trichophyton effractum* Sabouraud 1909

Cultures at first very similar to those of *T tonsurans* being crateriform when old the growth becomes very dry and the surface splits from the edge

*Trichophyton curru* Chalmers and Marshall 1914

It appears  
the type  
It forms  
pores are  
um resist  
in mono  
lucosides  
it liquefy

monkeys cats dogs and mice In man it gives rise to a  
of tinea capitis tropicalis

Chalmers 1918

resent  
aching  
r shaft

found in donkeys, and may infect man. The same or similar species are found parasitic on the horse and on some birds, and may also infect man.

**Ectotrichophyton ochraceum** Sabouraud, 1909

Synonym.—*Trichophyton ochraceum* Sabouraud 1909

Type Fectothrix, of animal origin, cultures somewhat similar to favus. On maltose and glucose agars the colonies are character-

25° C It is easily inoculated into guinea pigs

**Ectotrichophyton album** Sabouraud, 1907.

Synonym.—*Trichophyton album* Sabouraud 1907

The cultures are extremely like favus, but are generally less bulging, more deeply umbilicated and more regularly folded, the growth deepens in the medium, some white duvet present. Optimum temperature, 25° C. Can be inoculated into guinea pigs

**Ectotrichophyton discoides** Sabouraud, 1909

Synonym.—*Trichophyton discoides* Sabouraud 1909

Endo ectothrix, megalosporon, faviform. Somewhat similar to *E. album*, but the growth, which is almost a perfect disc, has a more flattened surface. There is often a central knob. The whole growth has a brownish yellowish colour, with a moist surface, somewhat resembling the non pigmented cultures of *Trichophyton violaceum*. It occurs in Egypt and in the Anglo Egyptian Sudan as described by one of us.

**Ectotrichophyton luxurians** Brault and Viguer, 1914

Isolated from cases of kerion in Algeria. Very rapid growth with faviform appearance.

**Ectotrichophyton (Microtrichophyton) Castellani and Chalmers, 1918**

Definition.—Ectotrichophyton with small spores 3-4 microns in diameter.

Type Species.—*Ectotrichophyton (Microtrichophyton) mentagrophytes* Robin, 1853

Classification.—The following species are known —

*E. mentagrophytes* Ch Robin, 1853

*E. farinulentum* Sabouraud, 1910

*E. persicolor* Sabouraud, 1910

*E. granulorum* Sabouraud, 1908

*E. lacticolor* Sabouraud, 1910

*E. radiolatum* Sabouraud, 1910

*E. felinum* R Blanchard 1895

*E. denticulatum* Sabouraud, 1910

Ectotrichophyton (Favotrichophyton) Castellani and Chalmers  
1918

Definition — Ectotrichophyton with the characters given above  
for Favotrichophyton

Type Species — *Ectotrichophyton discoides* Sabouraud 1909



FIG 506 — *Ectotrichophyton discoides* NINETEEN DAYS GROWTH ON SABOURAUD'S MALTOSE AGAR AT 32° C



FIG 507 — *Ectotrichophyton discoides* FORTY TWO DAYS GROWTH ON SABOURAUD'S MALTOSE AGAR AT 32° C

Classification — The Favotrichophyton species which are known are —

- E. verrucosum* Bodin 1902
- E. ochraceum* Sabouraud 1909
- E. album* Sabouraud 1909
- E. discoides* Sabouraud 1909
- E. luxurians* Brault and Viguer 1914

which may be differentiated as follows —

- A Condition of mycelium in hair not definitely stated —
  - I Young cultures white in colour and soon resembling those of *Achorion schoenleinii* but sunk into the medium — *Album*
- B Condition of mycelium in hair that of an ecto-endothrix —
  - II Cultures grey in colour humid with verrucose surface. — *verrucosum*

I

*Ectotrichophyton verrucosum* Bodin 1902

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vs cul  
rowth  
It is

grows better on agar without sugar than on sugar media. The cultures are of a pinkish reddish colour. The appearance of the colonies has been aptly compared by Adamson to the skin of a very ripe peach.

**Ectotrichophyton granulatum** Sabouraud 1908

**Synonym**—*Trichophyton gypsum* Bodin 1902 *pro parte* *Trichophyton granulatum* Sabouraud 1908

The growth on Sabouraud's agar is discoid, often umbilicated, powder surface of a white-yellowish colour with granular formations or prominences dotted all over.

This *Trichophyton* is found in the horse in which it produces a peculiar type of trichophytosis with extremely numerous very small patches. It has been observed in man in Italy by Dalla Favera.

**Ectotrichophyton lacticolor** Sabouraud 1910

**Synonym**—*Trichophyton gypsum* Bodin *pro parte* *Trichophyton lichen* Sabouraud 1910

The cultures are discoid, flattened with shallow furrows radiating from the centre. The colour is cream white with occasionally a slight yellow tinge. In old cultures there is abundant pleomorphic duvet. Can easily be inoculated in guinea pigs.

**Ectotrichophyton radiolatum** Sabouraud 1910

**Synonym**—*Trichophyton gypsum* Bodin *pro parte* *Trichophyton radiolatum* Sabouraud 1910

Isolated by Sabouraud from cases of kerion. Very similar to *E. mentagrophytes* from which it differs in culture by the colour being of less pure white and by the radiating projections being less marked or absent. After three to four weeks abundant white pleomorphic duvet appears.

**Ectotrichophyton felinum** R. Blanchard 1895

**Synonyms**—*Trichophyton niveum* Sabouraud *T. radians* Sabouraud, 1909, *T. felinum* R. Blanchard 1895

Endo ectothrix causes often a pustular ringworm of the body, less frequently attacks the hairs. In the pustules free spores and a few mycelial elements are seen. In the affected hairs the spores forming the parasitic sheath are of large dimensions, 7 to 9  $\mu$  in diameter. The growth on Sabouraud's medium is umbilicated with a white powdery surface and numerous radiating projections at the periphery.

**Pathogenicity**—This fungus is found in the cat, and probably

**Ectotrichophyton denticulatum** Sabouraud 1910

**Synonym**—*Trichophyton niveum* Sabouraud *pro parte* *T. denticulatum*. Almost identical with *E. felinum* but in cultures the radiating projections are much shorter and more pointed.

**Ectotrichophyton (Ectotrichophyton) Castellani and Chalmers**  
1918

**Definition**—*Ectotrichophyton* with large spores about 5 microns.

They may be differentiated as follows —

- A Grows best on agar without sugars—*Perricolor*  
 B Grow best on agar with sugars —  
 I Growth white elevated centre powdery surface radiating furrows  
 (a) Furrows well marked Pure white—*Mentagrophytes*  
 (b) Furrows poorly marked Not so white—*Radiolatum*  
 II Growth white discoid umbilicated but later knob in centre white powdery surface radiating furrows—*Farinulentum*  
 III Growth white yellowish dotted with granular projections—*Granulosum*  
 IV Growth cream white to yellowish not granular—*Lacticolor*  
 V Growth white with umbilicated centre with numerous radiating projections at periphery  
 (a) Projections well marked—*Felsineum*  
 (b) Projections poorly marked—*Denticulatum*

### *Ectotrichophyton mentagrophytes* Ch Robin 1853

Synonyms—*Microsporon mentagrophytes* Robin 1853 *Sporotrichum mentagrophytes* Saccardo 1886 *Trichophyton gypseum* Bodin 1902 *T. asteroides* Sabouraud 1909 *Trichophyton mentagrophytes* Robin 1853

Endo ectothrix, mycelial spores are mostly situated outside the cuticle of the hair while a few are found in the interior. The latter are 5 to 6  $\mu$  in size those outside forming the parasitic sheath are of very unequal size (2 to 11  $\mu$ )

This *Trichophyton* is of animal origin being found in horses cows dogs and perhaps pigs and sheep. In man it is pyogenic causing a type of trichophytic sycosis kerion and also a pustular type of tinea corporis.

The following five species are very closely allied to *E. mentagrophytes*—

### *Ectotrichophyton farinulentum* Sabouraud 1910

Synonyms.—*Trichophyton gypseum* Bodin 1902 *Trichophyton*

### *Ectotrichophyton persicolor* Sabouraud 1910

Synonym—*Trichophyton gypseum* Bodin 1902 *pro parte* *Trichophyton persicolor* Sabouraud 1910

Found by Sabouraud in cases of pustular ringworm of the palms of the hands and of the beard. In contrast to all other species of *Trichophyton* it



characteristic brick red colour, which generally disappears after repeated transplantations. The surface growth is whitish but more abundant than in the normal growth.

*Adonite*—Same as agar

Pathogen

cruris (p. 2)

nodules

of the body, in addition to the inguinal regions. It is capable of affecting the hair follicles. In one of our cases the fungus affected the hairs of the beard producing a typical 'kerion barbae'.

### Genus *Atrichophyton* Castellani and Chalmers, 1918

**Definition.**—*Trichophyton*eae with mycelium and spores present in the lesions and conidia on short stalks but they do not attack hairs.

**Type Species**—*Atrichophyton albiscicans* Nieuwenhuis, 1907

**Classification**—The following table will indicate the characters of the species—

#### A *Has been cultivated* —

- I Culture whitish with powdery surface—*Albiscicans*
- II Culture brownish mass with deep furrows—*Macfadyens*
- III Cultures pinkish with violet tinge—*Sanna*

#### B *Has not been cultivated* —

- I Spores are numerous and of various sizes—*Blanchardi*
- II Spores are few and about 4 microns in diameter—*Ceylonense*

### *Atrichophyton albiscicans* Nieuwenhuis 1907

**Synonym**—*Trichophyton albiscicans* Nieuwenhuis 1907

Discovered by Nieuwenhuis in tinea albigena. In fresh preparations from scrapings spores are absent the mycelial tubes are straight, occasionally showing a double contour they are often dichotomous. On Sabouraud's agar the growth is very slow, whitish with a powdery surface.

### *Atrichophyton blanchardi* Castellani 1905

**Synonym**—*Trichophyton sabouraudi* Castellani, 1905, *T. blanchardi* Castellani 1905

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ile  
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ial

Type Species — *Ectotrichophyton (Ectotrichophyton) megnini* R  
Blanchard 1895

Classification — The following species are known —

- E megnini* (R Blanchard 1895)
- E equinum* (Geddoelst 1902)
- E vinosum* (Sibouraud 1909)
- E no-to-formans* (Castellani 1912)

They may be distinguished as follows —

A Mycelial spores very large 8-9 in cross in diameter —

I Old

II Old

III Mycelial

I Su

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—EQUINUM

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grows better on agar without sugar than on sugar media. The cultures are of a pinkish reddish colour. The appearance of the colonies has been aptly compared by Adamson to the skin of a very ripe peach.

*Ectotrichophyton granulatum* Sabouraud 1910

Synonym.—*Trichophyton gypseum* Bodin *pro parte* *Trichophyton granulosum* Sabouraud 1910

The growth on Sabouraud's agar is discoid often umbilicated powdery surface of a white-yellowish colour with granular formations or prominences dotted all over.

This *Trichophyton* is found in the horse in which it produces a peculiar type of trichophytosis, with extremely numerous very small patches. It has been observed in man in Italy by Dalla Favera.

*Ectotrichophyton lacteolum* Sabouraud 1910

Synonym.—*Trichophyton gypsaceum* Bodin *pro parte* *Trichophyton lacteolum* Sabouraud 1910

The cultures are discoid flattened with shallow furrows radiating from the centre. The colour is cream white with occasionally a slight yellow tinge. In old cultures there is abundant pleomorphic duvet. Can easily be inoculated in Guinea pigs.

*Ectotrichophyton radiolatum* Sabouraud 1910

Synonym.—*Trichophyton gypseum* Bodin *pro parte* *Trichophyton radiolatum* Sabouraud 1910

Isolated by Sabouraud from cases of kerion. Very similar to *E. melleum* from which it differs in culture by the colour being of less pure white and by the radiating projections being less marked or absent. After three to four weeks abundant white pleomorphic duvet appears.

*Ectotrichophyton felineum* R. Blanchard 1903

Synonyms.—*Trichophyton melleum* Sabouraud *T. radians* Sabouraud 1909 *T. felineum* R. Blanchard 1903

puscular ringworm of the body.

In the pustules free spores and

in the affected hairs the spores

of large dimensions 7 to 9  $\mu$  in

diameter. The growth on Sabouraud's medium is umbilicated with a white powdery surface and numerous radiating projections at the periphery.

Pathogenicity.—This fungus is found in the cat, and probably also in horses, cattle, dogs, sheep and pigs. In man it causes a type of kerion celsi and also a type of vesiculo-pustular tinea corporis, called by Sabouraud *trichophytosis circinata distriiformis*, and 'herpes iris vesiculosus' by Bielt.

*Ectotrichophyton denticulatum* Sabouraud 1910

Synonym.—*Trichophyton mirum* Sabouraud *pro parte* *T. denticulatum*

Almost identical with *E. felineum* but in cultures the radiating projections are much shorter and more pointed.

*Ectotrichophyton* (*Ectotrichophyton*) Castellani and Chalmers 1918

Definition.—*Ectotrichophyton* with large spores about 57 microns.

Type Species—*Ectotrichophyton (Ectotrichophyton)* Blanchard 1895

Classification—The following species are known—

*E megnini* (R Blanchard 1895)

*E equinum* (Gedoelst 1907)

*E vinosum* (Sabouraud 1909)

*E nodiformans* (Castellani 1912)

They may be distinguished as follows—

A Mycelial spores very large 8-9 in rows in diameter—

I Old cultures pinkish—*Megnini*

II Old cultures deep wine red—*Vinosum*

B

III portion brick

*Ectotrichophyton megnini* R Blanchard 1895

Synonyms—*Trichophyton roseum* Bodin 1902 *T. rosaceum* Sabouraud 1902 *T. megnini* R Blanchard 1895

Endo ectothrix megalosporon downy culture type Mycelial

4-5-9  $\mu$  in diameter On

with a velvety appear

Duvet becomes very

in f wls and pigeons

may infect man causing a variety of tinea barbæ without suppuration

*Ectotrichophyton vinosum* Sabouraud 1909

Synonym—*Trichophyton vinosum* Sabouraud 1909

Endo ectothrix megalosporon of downy culture type Is very similar to *E megnini* but the colour of old cultures is of a deep wine red colour Abundant duvet Found by Sabouraud in a case of tinea circinata

*Ectotrichophyton equinum* Gedoelst 1907

Synonym—*Trichophyton equinum* Gedoelst 1907

type Mycelial

4 in breadth On

dant duvet later

with the medium

It is parasitic in

the horse and may infect man

*Ectotrichophyton nodiformans* Castellani 1912

Synonym—*Trichophyton nodiformans* Castellani 1912

Found in Ceylon in cases of dhobi itch and tinea barbæ not very abundant in the lesions On Sabouraud agar the growth is white with a powdery surface and a central small knob The growth deepens in the medium and the submerged portion has a

characteristic brick red colour which generally disappears after repeated transplantations. The surface growth is whitish.

*Glucose Agar*—Growth somewhat more abundant than in Sabouraud's agar. Colour of the surface and submerged growth

regions. It is capable of causing the fungus affected kerion barbae.

### Genus *Atrichophyton* Castellani and Chalmers 1918

**Definition**—*Trichophytoneae* with mycelium and spores present in the lesions and conidia on short stalks but they do not attack hairs.

**Type Species**—*Atrichophyton albiscicans* Nieuwenhuis 1907

**Classification**—The following table will indicate the characters of the species—

#### A *Has been cultivated* —

- I Culture whitish with powdery surface—*Albiscicans*
- II Culture brownish mass with deep furrows—*Macfadyens*
- III Cultures pinkish with violet tinge—*Isannas*

#### B *Has not been cultivated* —

- I Spores are numerous and of various sizes—*Blanchardi*
- II Spores are few and about 4 microns in diameter—*Ceylonense*

### *Atrichophyton albiscicans* Nieuwenhuis 1907

**Synonym**—*Trichophyton albiscicans* Nieuwenhuis 1907

Discovered by Nieuwenhuis in tinea albigena. In fresh preparations from scrapings spores are absent; the mycelial tubes are straight occasionally showing a double contour; they are often dichotomous. On Sabouraud's agar the growth is very slow, whitish with a powdery surface.

### *Atrichophyton blanchardi* Castellani 1905

**Synonym**—*Trichophyton sabouraudii* Castellani 1905 *T. blanchardi* Castellani 1905

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ile  
a  
ial

Type Species—*Ectotrichophyton (Ect. trichophyton) megnini* R Blanchard 1895

Classification—The following species are known—

*E megnini* (R Blanchard 1895)

*E equinum* (Gedoelst 1902)

*E vinosum* (Sabouraud 1909)

*E nodiformans* (Castellani 1912)

They may be distinguished as follows—

A 1895

I

port on brick

*Ectotrichophyton megnini* R Blanchard 1895

Synonym *Trichophyton megnini* R Blanchard 1895

celial

On

pear

and later pinkish or of a deep rose colour. Duvet becomes very abundant in old cultures. It is parasitic in fowls and pigeons may infect man causing a variety of tinea barbae without suppuration.

*Ectotrichophyton vinosum* Sabouraud 1909

Synonym—*Trichophyton vinosum* Sabouraud 1909

Endoecothrix megalosporon of downy culture type. Is very similar to *E megnini* but the colour of old cultures is of a deep wine red colour. Abundant duvet. Found by Sabouraud in a case of tinea circinata.

*Ectotrichophyton equinum* Gedoelst 1902

Synonym—*Trichophyton equinum* Gedoelst 1902

celial

On

later

and later portion of the growth which is in contact with the medium becomes yellowish and afterwards dark red. It is parasitic in the horse and may infect man.

*Ectotrichophyton nodiformans* Castellani 1912

Synonym—*Trichophyton nodiformans* Castellani 1912

Found in Ceylon in cases of dhobi itch and tinea barbae not very abundant in the lesions. On Sabouraud's agar the growth is white with a powdery surface and a central small knob. The growth deepens in the medium and the submerged portion has a

grows better on agar without sugar than on sugar media. The cultures are of a pinkish reddish colour. The appearance of the colonies has been aptly compared by Adamson to the skin of a very ripe peach.

*Ectotrichophyton granulatum* Sabouraud, 1908

= Bodin, 1902 *pro parte* *Trichophyton*

is discoid often umbilicated, powdery with granular formations or prominences

This *Trichophyton* is found in the horse in which it produces a peculiar type of trichophytosis with extremely numerous very small patches. It has been observed in man in Italy by Dalla Favera.

*Ectotrichophyton lacticolor* Sabouraud, 1910

**Synonym.**—*Trichophyton gypsum* Bodin *pro parte* *Trichophyton lichenolor* Sabouraud 1910

The cultures are discoid flattened with shallow furrows radiating from the centre. The colour is cream white with occasionally a slight yellow tinge. In old cultures there is abundant pleomorphic duvet. Can easily be inoculated in guinea pigs.

*Ectotrichophyton radiolatum* Sabouraud 1910

**Synonym.**—*Trichophyton gypsum* Bodin *pro parte* *Trichophyton radiolatum* Sabouraud 1910

Isolated by Sabouraud from cases of kerion. Very similar to *E. mentis-grophytes* from which it differs in culture by the colour being of less pure white and by the radiating projections being less marked or absent. After three to four weeks abundant white pleomorphic duvet appears.

*Ectotrichophyton felineum* R. Blanchard, 1895

**Synonyms.**—*Trichophyton mucum* Sabouraud, *T. radians* Sabouraud, *T. felinum* R. Blanchard 1895

the body,  
cores and  
the spores  
0.9  $\mu$  in  
bilicated  
projections

at the periphery

**Pathogenicity.**—This fungus is found in the cat, and probably also in horses, cattle, dogs, sheep, and pigs. In man it causes a type of kerion celsi and also a type of vesiculo-pustular tinea corporis, called by Sabouraud 'trichophytosis circinata disidriiformis,' and 'herpes iris vesiculosus' by Bielt.

*Ectotrichophyton denticulatum* Sabouraud, 1910

**Synonym.**—*Trichophyton niveum* Sabouraud *pro parte* *T. denticulatum*. Almost identical with *E. felineum*, but in cultures the radiating projections are much shorter and more pointed.

*Ectotrichophyton* (*Ectotrichophyton*) Castellani and Chalmers, 1918

**Definition.**—*Ectotrichophyton* with large spores about 5.7 microns.

spores are shed without forming a filament by their union and are of various sizes. All attempts at cultivation have failed. It is the cause of tinea Sabouraudi tropicalis.

**Atrichophyton viannai** de Mello 1917

**Synonym** — *Trichophyton viannai* de Mello 1917 Found by F. de Mello in a case of tinea corporis. Colonies on Sabouraud's maltose agar pinkish with often a violet tinge.

**Atrichophyton ceylonense** Castellani 1908

*Trichophyton* Tr. 22 " " " " " "

**Atrichophyton macfadyeni**

Castellani 1905

**Synonym** — *Trichophyton macfadyeni* Castellani 1905

Found by Castellani in some cases of tropical tinea corporis. In fresh preparations mycelium and spores are rather of small dimensions. The mycelial tubes are regularly shaped, do not show swellings and are about  $2\frac{1}{2} \mu$  in breadth. The free spores are very numerous and present a peculiar void shape, the maximum diameter being  $2\frac{1}{2}$  to  $3\frac{1}{2} \mu$ . In stained preparations the spores present a bipolar staining. The fungus grows with difficulty on the rare occasions when the inoculations are successful, the growth is very slow, the colonies coalesce forming a brownish mass with deep furrows and deeply rooted in the medium.



1 to 508 — *Atrichophyton macfadyeni* CASTELLANI (Stained with fuchsin)

When grown on glucose agar from scales it slowly formed a somewhat crinkled growth of whitish colour. An interesting characteristic of this fungus is that apparently it does not become

INCERTÆ SEDIS

**Trichophyton baleaneum** Castellani 1916

Found in cases of peculiar condition of the scalp in the Balkans which clinically resembled more a diffuse type of severe *psoriasis* than a trichophytic affection.

When grown on glucose agar from scales it slowly formed a somewhat crinkled growth of whitish colour. An interesting characteristic of this fungus is that apparently it does not become



characteristic brick red colour, which generally disappears after repeated transplantations. The surface growth is whitish.  
*Glucose Agar*—Growth somewhat more abundant than in red growth.

pe of tinca

**Genus *Atrichophyton* Castellani and Chalmers 1918**

**Definition**—Trichophytonæ with mycelium and spores present in the lesions and conidia on short stalks but they do not attack hairs.

**Type Species**—*Atrichophyton albiseicans* Nieuwenhuis 1907

**Classification**—The following table will indicate the characters of the species—

**A Has been cultivated —**

- I Culture whitish with powdery surface—*Albiseicans*
- II Culture brownish mass with deep furrows—*Macfadyeni*
- III Cultures pinkish with violet tinge—*Sannasi*

**B Has not been cultivated —**

- I Spores are numerous and of various sizes—*Blanchardi*
- II Spores are few and about 4 microns in diameter—*Ceylonense*

***Atrichophyton albiseicans* Nieuwenhuis 1907**

1 prepara  
 tubes are  
 are often  
 ry slow

whitish with a powdery surface

***Atrichophyton blanchardi* Castellani 1905**

**Synonym**—*Trichophyton sabouraudii* Castellani 1905 *T. blanchardi* Castellani 1905

fresh  
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 v a  
 chial

**Type Species.**—*Ectotrichophyton (Ectotrichophyton) megnini* R Blanchard, 1895

**Classification.**—The following species are known —

- E megnini* (R Blanchard, 1895)
- E equinum* (Gedoelst, 1902).
- E vinosum* (Sabouraud 1909)
- E nodiformans* (Castellani, 1912).

They may be distinguished as follows —

- A Mycelial spores very large 8-9 microns in diameter —
  - I Old cultures pinkish—*Megnini*
  - II Old cultures deep wine red—*vinosum*
- B

portion brick

### *Ectotrichophyton megnini* R Blanchard 1895

**Synonyms.**—*Trichophyton roseum* Bodin, 1902, *T. rosaceum* Sabouraud, 1902, *T megnini* R Blanchard, 1895

Endo ectothrix, megalosporon, downy culture type Mycelial spores found in the hairs are very large, 8 to 9  $\mu$  in diameter On maltose agar the growth is at first white, with a velvety appearance, later, pinkish, or of a deep rose colour Duvet becomes very abundant in old cultures It is parasitic in fowls and pigeons, may infect man, causing a variety of tinea barbæ without suppuration

### *Ectotrichophyton vinosum* Sabouraud, 1909

**Synonym**—*Trichophyton vinosum* Sabouraud, 1909

Endo ectothrix, megalosporon, of downy culture type Is very similar to *E megnini*, but the colour of old cultures is of a deep wine red colour Abundant duvet Found by Sabouraud in a case of tinea circinata

### *Ectotrichophyton equinum* Gedoelst, 1902.

**Synonym.**—*Trichophyton equinum* Gedoelst 1902

Endo ectothrix, megalosporon downy culture type Mycelial spores of oval shape, 4 to 6  $\mu$  in length, 2 to 4  $\mu$  in breadth On maltose agar the growth is orbicular, with abundant duvet, later on, the portion of the growth which is in contact with the medium becomes yellowish and afterwards dark red It is parasitic in the horse, and may infect man

### *Ectotrichophyton nodiformans* Castellani 1912

**Synonym**—*Trichophyton nodiformans* Castellani 1912

Found in Ceylon in cases of dhobi itch and tinea barbæ, not very abundant in the lesions On Sabouraud's agar the growth is white, with a powdery surface and a central small knob The growth deepens in the medium and the submerged portion has a

TABLE SHOWING BIOCHEMICAL CHARACTERS OF *T. BALCAEUM*.

Molality Gram	Gelatin			Serum			Lactin Milk			Lactose			Saccha- rose			Dulcitol			Mannite			Glucose			Maltose			
	Day	8	12	Day	4	8	12	Day	4	8	12	Day	4	8	12	Day	4	8	12	Day	4	8	12	Day	4	8	12	
<i>Trichophyton balcaenum</i>	+	+	+	+	+	+	+	AsC	AsC	AsC	AsC	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+

Day	Dextrin			Raffinose			Arabinose			Adonite			Inulin			Starch			Salicin			L-rulose			Galactose			Glycerine		
	4	8	12	4	8	12	4	8	12	4	8	12	4	8	12	4	8	12	4	8	12	4	8	12	4	8	12	4	8	12
<i>Trichophyton balcaenum</i>	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+

Abbreviations used in the Table—A=acid, G=gas, C=clot; s=slight, vs=very slight, O=negative result—vir, non-pro-  
duction of acid or gas in sugar media, non liquefaction of gelatine or serum, as the case may be. + =positive result.



been compared by some authorities to the spindles of the *Trichophytons* and *Microsporons* but they are slenderer and not septate. The French authors call these formations *chandeliers faviques* on account of their shape somewhat resembling a candlestick.

2 *Favus Yellow Bodies*—The protoplasm of some filaments collects itself at the terminal ends the filaments becoming much thinner and terminating in roundish or oval bodies which must be considered to be terminal chlamydo-spores. It is to be noted that in the typical *Achorions* of human origin conidia bearing hyphae are not seen while these are present in the *Achorions* of animal origin in which separate spindles may be present.

**Media**—*Achorions* grow well on all the usual sugar media especially Sabouraud's agar and glucose agar. They generally liquefy gelatine fairly rapidly.

**Pleomorphism**—Very common in all *Achorions*. If a culture becomes pleomorphic it is impossible to make it revert to the original type.

**Transmission**—In the case of *Achorions* of human origin infection takes place from man to man in the case of *Achorions* of animal origin infection takes place from the lower animals. It is not impossible that *Achorions* may live saprophytically in nature.

TABLE OF ACHORIONS

Genus.	Spec. s.	
Achor on Remak 1845	Of human origin (typical) } A. schoenleini Lebert 1845	
	Of animal origin (non typical) } A. quinckeanum Zopf 1890	
		A. gypseum Bodin 1907
		A. arloingi Blanchard 1891

These may be recognized as follows—

- A. Whitish yellow cerebriform colonies—*Schoenleini*
- B. White downy colonies—*Quinckeanum*
- C. Yellowish colonies—*Gypseum*

*A. arloingi* has not been properly described.

#### *Achorion schoenleini* Lebert 1845

**Synonyms**—*Oidium schoenleini* Lebert 1845 *O. porriginis* Montague *Oospora porriginis* Saccardo 1886 *Oidium schoenleini* Zopf 1890

Causes the well known affection called *favus* which is characterized by the presence of peculiar disc shaped crust formations called *scutula* of a sulphur yellow colour and emitting an offensive odour which has been compared to the smell of mice's urine.

The fungus may infect the hairs or glabrous parts of the body it may attack the nails. In the hairs the mycelium is very abundant the segments being comparatively long. Sometimes the mycelial threads divide into three or four branches each of which terminates in a single row of roundish spores. This is known as *favic tarsus*.

pleomorphic even after very numerous transplantations. Gelatine is rapidly liquefied. It does not form gas in any carbohydrate medium.

Microscopically the fungus shows features intermediate between a trichophyton *sensu lato*, a microsporium and an achorion.



FIGS 509 AND 510—CULTURES OF *Trichophyton balcanicum* CASTELLANI GLUCOSE AGAR



FIG 511—MICROSCOPICAL APPEARANCES OF *Trichophyton balcanicum* CASTELLANI HANGING DROP CULTURE

### Genus Achorion Remak 1845

**Definition**—Trichophytonæ with mycelial filaments and spores in the lesions in cultures conical bearing hyphæ present with spores situate laterally and apically. Fusiform bodies in cultures in the form of swollen claviform ends of filaments. Yellow faviæ scutula present in lesions.

pa  
an

form the well known sulphur coloured *scutula* which always develop round a hair. In the hairs the mycelial tubes are frequently trichotomous and tetrachotomous forming structures which have been compared in appearance to the skeleton of the human foot and called *favus tarsi*.

**Cultures**—Sabouraud has noted that in hanging drop cultures the spores sometimes develop very slowly sometimes rapidly. When the development is slow there is formation of numerous chlamydospores of various sizes with a double contour membrane. When the development is rapid one notes that the mycelium ramifies quickly in every direction with presence of very few or no chlamydospores. The following structures may be noted:

1. **Claviform Bodies**—The terminal portion of some filaments becomes swollen and claviform. These claviform filaments have

**Achorion quinekeanum** Zopf 1890

The fungus appears in the lesions in the shape of numerous  
 m. cel al fil — ated in so many small segments  
 rows readily on Sabouraud's agar

This fungus botanically holds an intermediate position between  
 the typical *Achorions* and the *Microsporons* and *Trichophyton*  
 conidia bearing hyphæ of the type *Acladium* being present. It  
 produces however typical favus lesions with scutula.

**Pathogenicity**—Is the cause of favus in mice and may occa-  
 sionally infect man in whom too it induces typical favus. It can  
 be inoculated into guinea pigs.

**Achorion gypseum** Bodin 1907

Found by Bodin in 1907 in a typical case of favus. On Sabour-  
 aud's agar the growth which is roundish presents a little white  
 duvet in the centre while the rest has a reddish  
 colour. Old cu-

dant white duvet  
 closely allied to *A. quinekeanum* and *Microsporons* but gives rise  
 to typical favus with scutula when inoculated in mice and guinea  
 pigs. The inoculation of pleomorphic cultures does not cause any  
 eruption.

**Achorion arisingsi** R. Blanchard 1891

**Synonym** — *A. quinekeanum*  
 Incomplete m a human trichophytic  
 like eruptio Is said to be inoculable  
 into mice r

**Genus Lophophyton** Matruchot and Dessonville 1899

M. cel al filaments either tortuous very thin or thick with granular  
 protoplasm. No spores present. One species only.

**Lophophyton gallinæ** Mégnin 1881

**Synonym** — *A. quinekeanum*

Favus lesions  
 erythematous

**Genus Epidermophyton** Lang 1879 *emendavit* Sabouraud 1907

**Definition** —  
 present in the m  
 the cultures s  
 in the superficial layers of the epidermis

**Type Species** — *Epidermophyton cruris* Castellani 1905

**Remarks**—The fungi belonging to this genus which has been  
 investigated by Sabouraud and Castellani grow superficially on the

Cultures—The fungus is easily cultivated on various media. On Sabouraud's agar and on glucose agar the growth when completely developed is convoluted or cerebriform and somewhat bulging.

After a  
duvet is

four days  
to twenty

days

By inoculation of pure cultures of the fungus favus lesions are produced in man, dogs, mice, rabbits, and fowls. The inoculation in guinea pigs does not give rise to typical favus lesions with scutula, but to circinate trichophytic like lesions.



FIG. 512.—SO-CALLED YELLOW BODIES IN CULTURES OF *Achorion schoenleini* LEBERT (After Bodin)



FIG. 513.—CLAVIFORM BODIES IN CULTURES OF *Achorion schoenleini* LEBERT

Reproduction—This takes place—

- 1 By sprouting
- 2 By elongated funniform structures analogous to

... to be chlamydo-spores (see also general remarks on ...)



of tinea cruris. It is not inoculable into guinea pigs. Attempts at reproducing the eruption in man by inoculating pure cultures have also failed.

#### **Epidermophyton perneti Castellani, 1907**

This fungus has been described by Pernet. It differs from Sabouraud's agar and which is generally

#### **Epidermophyton rubrum Castellani, 1909**

**Synonym.**—*Trichophyton purpurium* Bang, 1910

This fungus was described by Castellani in Ceylon in 1909 and by Bang in France in 1910. On Sabouraud's agar the growth begins to appear four to six days after inoculation as a raised red spot, which gradually enlarges. At complete development the growth is of a deep red colour, either with a central knob or crater form, and is partly covered with a white, delicate duvet. In old cultures the white duvet is much more abundant and thicker, and may hide the red pigmentation almost completely.

On glucose agar (4 per cent) which is the best medium for this fungus, the growth is of a very deep blood red colour, and the red pigmentation may spread to portions of the medium itself. In old cultures abundant white—occasionally white-greenish—duvet is present. This may hide the pigmentation, but, scraping out the duvet, the red pigmentation will be found to be still well marked. On ordinary agar and glycerine agar the fungus grows fairly well, but there is no red pigmentation.

#### **Genus Endodermophyton Castellani, 1909**

**Definition.**—Trichophytonæ with mycelial filaments and spores in the lesions, but no conidial filaments in cultures. Pluriseptate  
ers

ized  
the  
epidermis, forming an interlacing felt of mycelia, which detaches

minutes, must be placed in glucose broth tubes, one scale in each tube. Most of the tubes become contaminated with bacteria, but in those which remain clear, after a time (five to ten days) a few

been isolated from human lesions except the *Epidermophyton* discovered by Pinoy in monkeys

TABLE OF EPIDERMOPHYTONS

Genus	Sp. cat.
<i>Epidermophyton</i> Lang 1879 <i>emendavst</i>	<i>E. cruris</i> Castellani 1905
Sabouraud 1907	<i>E. pernici</i> Castellani 1907
	<i>E. rubrum</i> Castellani 1907

These species may be recognized by their growths on Sabouraud's agar —

- A Colour peculiar yellow—*Cruris*
- B Colour pinkish—*Pernici*
- C Colour deep red—*Rubrum*

For *E. similis* Pinoy 1911 we have created the genus *Pinoyella*

#### *Epidermophyton cruris* Castellani 1905

**Synonyms**—*Trichophyton cruris* Castellani 1905 *Epidermophyton inguinalis* Sabouraud 1907 *T. castellani* Brooke 1908

Found in cases of tinea cruris in Ceylon by Castellani and in France by Sabouraud. The fungus is very abundant in recent cases extremely scarce in old ones. The mycelial tubes in recent cases are generally straight have often a double contour and the segments are somewhat rectangular their breadth being  $3\frac{1}{2}$  to  $4\frac{1}{2}$   $\mu$ . Branching is not rare. The spores are rather large (4 to 7  $\mu$ ) roundish and have generally a double contour they do not collect in clusters. In chronic cases degeneration forms of the fungus are met with the mycelium may be banana shaped may show several constrictions or long strings of ovoid elements may be seen.

This *Epidermophyton* grows well but rather slowly on Sabouraud's agar. The growth begins to be visible after four to eight days the colonies being at first of a peculiar yellow colour lemon yellowish or orange-yellowish occasionally with a greenish tinge. Later they become white with pulverulent surface and may be acuminate or crateriform. Pleomorphism with abundant white duvet develops quickly.

This fungus in Ceylon is the commonest species found in cases

recently four, further investigation having shown that the term he used for one species (*concentricum*) covered more than one species

FIG. 514.

y abundant in the lesions,  
celial threads with mycelial  
or comes but as --

the mycelial articles, which are

$3\frac{1}{2}$   $\mu$  in breadth, will be seen to have a double contour. *Aspergillus*



FIG. 514.—*Endodermophyton tropicale* CASTELLANI' GLUCOSE AGAR CULTURE



FIG. 515.—*Endodermophyton tropicale* CASTELLANI' OLD GLUCOSE AGAR CULTURE.

fructifications, described by so many authors when present are due to contamination.

*Glucose Agar (4 per cent)*.—Growth abundant, surface cerebriform or crinkled. The growth and the medium show a slight amber colour, which later on may become of much deeper hue. Duvet as a rule absent but in old cultures which have been transplanted many times and are degenerating some very scarce short, whitish duvet may appear.

*Sabouraud Agar*.—Growth comparatively scanty, whitish-grey, mostly submerged. The colonies are whitish, have generally a small central knob,

delicate, short white filaments will be seen originating from the scale. The growth slowly increases until after three to four weeks it takes the appearance of a small white, fluffy mass with a dark

The fungi grow much more abundantly on glucose agar, + plus on other media

al filaments  
reduction is  
but further

l than in the  
old cultures  
duvet

TABLE SHOWING ENDODERMOPHYTONS FOUND IN MAN IN ORDER OF FREQUENCY

- E. tropicale* Castellani
- E. indicum* Castellani
- E. concentricum* Blanchard
- E. mansoni* Castellani

These may be recognized as follows —

- A Glucose agar cultures amber coloured no duvet or only slight—*Tropicale*
- B Glucose agar cultures deep red —
  - I Causes *Tinea imbricata*—*Indicum*
  - II Causes *Tinea intersepta*—*Castellani*
- C Glucose agar cultures after a time black —
  - I Pigmentation fairly slow—*Concentricum*
  - II Pigmentation very rapid—*Mansoni*

*Endodermophyton tropicale* Castellani 1914

Remarks—Manson in 1872 described a trichophyton like organism in the squamæ of *tinea imbricata*, with the laboratory attempts at a cultivation did not succeed  
hyton was being years

saprophytes or contact has succeeded in growing causing the disease

form of *tinea imbricata*, and that from the scales of the eruption, experimentally induced, the same fungus is recoverable. For further details see Chapter XCII on *Tinea Imbricata* (p 2509)

**Endodermophyton indicum Castellani, 1911.**

cat  
ide

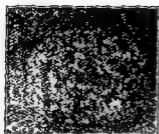


FIG. 518 — PATCH OF EXPERIMENTAL *TINEA IMBRICATA* IN A SINGHALESE BOY OBTAINED BY INOCULATING A CULTURE OF *Endodermophyton indicum*.



FIG. 519 — *Endodermophyton indicum* CASTELLANI HANGING DROP CULTURE

solid media, when the growth takes place in the dark, about 80° to 85° F., without rubber caps on the tubes, and is between fifteen and twenty-one days old, are as follows —

*Glucose Agar* (4 per cent) — Growth fairly abundant with surface somewhat convoluted or furrowed. Portions of the growth often the central

duvet  
y surface covered by  
covered by short white  
but the surface  
white duvet is  
more abundant

*Lactose Agar* — Knobby surface covered by snow white duvet

*Nutrose Agar* — Yellowish crinkled surface, short white duvet present

*Levulose Agar* — Scanty growth, yellow or orange, scarce; very short white duvet present

*Galactose Agar* — Fairly abundant, surface convoluted with abundant

and never show any duvet. The submerged portion is very firmly embedded and often presents projections deepening in the medium. Colour of the

the colonies

! No duvet  
glucose but  
colour No

duvet

*Saccharose Agar (4 per cent)* — Growth rather scanty similar to Sabouraud. Duvet absent.

*Maltose Agar (4 per cent)* — Very slow growth. Separate young colonies have a central knob, they coalesce later into a knobby mass.



FIG 516 — *Endodermophyton tropicale* CASTELLANI, AGAR CULTURE



FIG 517 — *Endodermophyton tropicale* CASTELLANI MALTOSE AGAR CULTURE

ures (Sabouraud's  
Reproduction is  
hing taking place  
common type of  
that the inocula-  
tions into human beings of cultures of the fungus reproduce a typical

times inoculated. CASTELLANI is the only one who

caps are used both *E. indicum* and *E. tropicalis* may assume a bright red colour. If however subcultures are made from these using tubes without rubber caps the fungi again show the characters given above.

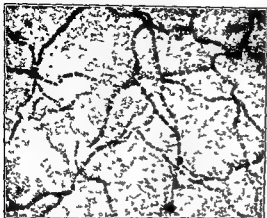


FIG 522—*Endodermophyton indicum* CASTELLANI IN THE SCALES

Castellani has succeeded in experimentally reproducing the disease by inoculating coolies, who had volunteered with pure cultures of the fungus. The skin was first scarified with a sterile knife, then a certain amount of a pure agar culture of *E. indicum* was well rubbed in. After fifteen to twenty one days the first signs of the eruption appeared and the typical patches of *tinea imbricata* developed. From the scales of the experimental cases a fungus was grown absolutely identical with the strain of *E. indicum* with which the individuals had been inoculated.

*Endodermophyton castellanii*  
Perry 1907

Found by Castellani in cases of *tinea intersecta*. In the scales the mycelium is fairly abundant.

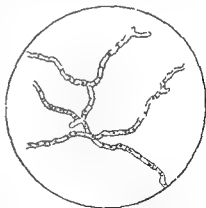
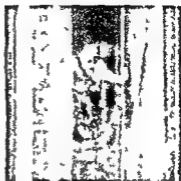


FIG 523—*Endodermophyton castellanii* PERRY

roundish refringent dots, one at each extremity. Has been potassé contour has two

separated

come

FIG 520—*Endodermophyton indicum*  
CASTELLANI AGAR CULTUREFIG 521—*Endodermophyton indicum*  
CASTELLANI GLUCOSE  
AGAR CULTURECOMPARISON BETWEEN THE CULTURAL CHARACTERS OF *Endodermophyton tropicale* AND OF *Endodermophyton indicum*

Media	<i>E. tropicale</i>	<i>E. indicum</i>
Glucose agar	Amber colour duvet absent in young cultures	Deep orange at times pinkish or red very short white delicate duvet often present
Sabouraud agar	Growth scanty mostly sub merged grey whitish duvet absent	Surface growth more abundant powdery white
Agar	Scanty, mostly submerged similar to Sabouraud	Fairly abundant knobby well marked snow white
Glycerine agar		
	Sabouraud agar no duvet	present

The above characters are based upon the appearance of cultures kept in the dark at a temperature 80° to 90° F and without rubber caps. If any of these conditions are altered the cultural characters are changed. If rubber



## FAMILY ASPERGILI ACEÆ

**Definition**—Aspergillales with compact peridium small sessile closed perithecia

**Type Genus**—*Aspergillus* Micheli 1729

**Classification**.—The genera of the Aspergillaceæ can be recognized as follows—

A *Spores unicellular*—

I Perithecium beaked—*Microascus*

II Perithecium not beaked—

(a) Perithecia with appendages—*Cephalotheca*

(b) Perithecia without appendages—

1 Conidiophores absent—*Thelasia*

2 Conidiophores present—

(A) Conidia solitary—*Aphanoascus*

(B) Conidia in chains—

(i) Conidiophores simple—*Emericella*

(ii) Conidiophores enlarged apically and bearing sterigmata—

(1) Sterigmata simple—*Aspergillus*

(2) Sterigmata branched—*Sterigmatocystis*

(iii) Conidiophores branched—

(1) Sympodially branched—*Eurotopsis*

(2) Bushy branched—

(a) In bundles perithecia stalked—*Penicillioopsis*

(b) Not in bundles perithecia sessile—*Penicillium*

B *Spores bicellular*—*Testudina*

We are concerned with the genera *Penicillium*, *Aspergillus*, and *Sterigmatocystis*, of which the following species are parasitic in man

<i>Penicillium</i> Link 1809	{	<i>P crustaceum</i> Linnaeus 1761
		<i>P minimum</i> Siebenmann 1889
		<i>P barba</i> Castellani 1907
		<i>P montojas</i> Castellani 1907
		<i>P pruriosum</i> Salisbury
		<i>P brevicaulis</i> var <i>hominis</i> Brumpt and Langeron 1910
<i>Sterigmatocystis</i> Cramer 1869	{	<i>S antiaustriaca</i> Cramer 1859
		<i>S nidulans</i> Eidam 1883
<i>Aspergillus</i> Micheli 1725	{	<i>A fumigatus</i> Fresenius 1775
		<i>A flavus</i> Link 1791
		<i>A bronchialis</i> Blumentritt 1901
		<i>A nigrescens</i> Roben 1851
		<i>A repens</i> De Bary 1870
		<i>A malignus</i> Lindt 1889
		<i>A pictor</i> R Blanchard 1895
		<i>A barba</i> Castellani 1907
		<i>A bouffardi</i> Brumpt 1905
		<i>A herbariorum</i> Wiggers 1780
		<i>A fonteynonis</i> Guéguen 1909

cultivated only on one occasion the cultures being apparently similar to those of *E. indicum*. It is the cause of tinea intersecta.

**Endodermophyton concentricum** Blanchard 1901

Cultures on glucose agar are at first amber colour but after four to eight weeks become jet black and covered with abundant duvet.

**Endodermophyton mansonii** Castellani 1914

Portions of the growth are black from the very first scanty duvet.

**Genus Pinoyella** Castellani and Chalmers 1908

**Definition**—Trichophytoneæ with mycelial filaments and spores in the lesions and in cultures conidial bearing hyphæ with the spores situate laterally only.

**Type and only Species**—*Pinoyella similis* (Pinoy 1911) *Epidermophyton similis* Pinoy 1911

Discovered by Pinoy in a trichophytic like eruption observed in a monkey. On Sabouraud's agar the growth is at first yellowish

**Genus Montoyella** Castellani 1907

**Definition**—Trichophytoneæ with mycelial filaments and spores in the lesions in cultures conidial bearing hyphæ with only terminal spores.

**Remarks**—Temporary genus. Two kinds of mycelial threads, some slender ramified septate others much thicker having

A Cultures black—*Nigra*

B Cultures whitish or greenish—*Bodini*

**Montoyella nigra** Castellani 1907

Temporary species. Colonies on maltose agar are black. If glycerine agar is used the medium takes a black colour. This species discovered by Montoya is common in black pinta.

**Montoyella bodini** Castellani 1907

Temporary species. Colonies whitish or greenish.

**Penicillium brevicaulis** var **hominis** Brumpt and Langeron 1910

Found by Brumpt and Langeron in two cases of onychomycosis. In the lesions septate mycelial threads were seen 2 to 10  $\mu$  diameter and large groups of chlamydo-spores generally terminally 10 to 30  $\mu$  in diameter. The fungus is easily cultivated on Sabouraud's agar and other sugar media also on potatoes and carrot. Optimum temperature 25°C but grows well also at 37°C. Conidia spherical or occasionally ovoid of a chocolate colour.



FIG 527—*Penicillium brevicaulis* VAR *hominis* BRUMPT AND LANGERON  
(After Brumpt)



FIG 528—*Penicillium brevicaulis* VAR *hominis* BRUMPT AND LANGERON  
(Preparation from the diseased nails after Brumpt)

**Penicillium barbæ** Castellani 1907

Found by us growing on the beard of natives of equatorial Africa and in natives of Ceylon.

**Penicillium pruriens** Salisbury

Doubtful species found by Salisbury in the vaginal mucus of a woman suffering from intense vaginal pruritus.

**Genus Aspergillus** Micheli 1725

The conidiophore hyphæ are not ramified and terminate into ovoid or roundish formations which support numerous claviform elements (sterigmata) each of which supports a chain of roundish conidia.



FIG 529—**ASPERGILLUS**  
**FRUCTIFICATION**  
(After Brumpt)

*Aspergilli* are generally saprophytes but they may become parasites. They are easily grown on acid media liquid or solid and also on alkaline media. Iron and manganese have a favourable influence on the growth and sporulation of these fungi.

A medium much used is Raulin's liquid the formula of which is: Cane sugar 70 grammes tartaric acid 4 grammes ammonium nitrate 4 grammes ammonium phosphate 0.60 gramme potassium carbonate 0.60 gramme magnesium carbonate 0.40 gramme ammonium sulphate 0.25 gramme zinc sulphate 0.07 gramme ferrous sulphate 0.07 gramme potassium silicate 0.07 gramme.

Fungi of the genus *Aspergillus* when growing parasitically in the tissues often lose their characteristics, the typical fructifications



Besta have isolated toxic products soluble in ether and alcohol which act on the muscular and nervous system of dogs and rabbits (See also remarks on *Aspergillomycoses*, p 1031)

*Aspergillus flavus* De Bary, 1870

Synonyms — *Monilia aurea* Gmelin, 1791, *Eurotium flavum* De Bary, 1870, *Aspergillus flavescens* Wreden 1874

Mycelium colourless, conidiophore hyphæ terminate in roundish formations of a gold yellow colour. The conidia are dark yellow roundish 5 to 7  $\mu$  in diameter, with a surface showing numerous minute mammillary prominences. Found by several observers (Wreden, Siebenmann, etc.) in the ear. Optimum temperature 37° C —

*Aspergillus bronchialis* Blumentritt, 1901

by Blumentritt. It has not yet been found as a saprophyte

*Aspergillus fontoyonti* Gueguen 1909

*Aspergillus malignus* Lindt, 1889

Synonym — *Eurotium malignum* Lindt 1889

Colourless mycelium composed of short articles. Conidiophore hyphæ erected, terminating in pyriform formations, 22 to 24  $\mu$  in



FIG 532 — *Aspergillus malignus*  
LINDT  
(After Lindt)



FIG 533 — *Aspergillus repens*  
DE BARY  
(After Siebenmann)

diameter, on which are situated the sterigmata, which are 10  $\mu$  long and 4 to 5  $\mu$  in breadth. Conidia roundish, of a greenish bluish colour

are absent and only mycelial threads and roundish or oval yeast like bodies are seen

The various species may be differentiated with difficulty as follows or cannot be differentiated —

A Green species —

I Spores 5 microns and more · Can hardly be differentiated—

Repens Flavus

“ “

**Aspergillus fumigatus Fresenius 1875**

The other ...

rounded the breadth varying between 2 and 3  $\mu$ . The conidiphore hyphæ are much thicker than the mycelial tubes being on the average about 5  $\mu$  in breadth. The sterigmata which are situated very close together are 6  $\mu$  long the conidia are roundish 2.5 to 3  $\mu$  in diameter smooth colourless. Optimum temperature 37° C the growth stops below 20° or above 55° C



FIG 530 — *Aspergillus fumigatus*  
FRESENIUS



FIG 531 — *Aspergillus bronchialis* BLUMENTRIIT  
(After Blumentritt)

**Pathogenicity** — This *Aspergillus* is the species most frequently found in man giving rise to an aspergillosis of various organs. The antiseptic effects and action of cultures of Genn and

*Aspergillus bouffardi* Brumpt 1905

Found by Bouffard in a case of black m —  
described by Brumpt

Chlamydospores present  
cultivation did not succeed

*Aspergillus barbæ* Castellani 1907

Found by us in natives of Uganda and in natives of Ceylon Conidia  
spherical 4 to 5  $\mu$  of a brownish colour

*Aspergillus nigrescens* Robin 1889

Doubtful species which according to Wæienfeld causes a faviform eruption.

Genus *Sterigmatocystis* Cramer 1859

**Definition**—Aspergillales with conidiophores terminating in  
roundish or ovoid formations on which are situated short cylindrical  
sterigmata—*primary sterigmata*—surmounted by similar elements—  
*secondary sterigmata* Each of the secondary sterigmata supports  
a chain of roundish conidia

**Remarks**—The genus was created by Cramer for a fungus found  
in a man's ear

**Type Species**—*Sterigmatocystis antacustica* Cramer 1859

**Classification**—The two species of importance to us can be  
recognized as follows—

- A Young conidial forms green later strongly grey to brown Conidia  
small 3 microns in diameter—*Nidulans*
- B Conidial forms blackish brown Conidia small 2.5 microns in  
diameter—*Antacustica*

*Sterigmatocystis antacustica* Cramer 1859

**Synonyms**

*nigrum* De B  
Wreden 187

Primary and secondary sterigmata erected conidiophores  
hyphæ more than 1 millimetre in length (3.5 to 4.5) Conidia  
globular 2.5  $\mu$  in diameter provided with a membrane of a brownish  
colour Commonly found in decaying organic substances It was  
first observed in man by Cramer who observed it in the ear of a  
deaf patient Later it was observed by Furbringer and others in  
mycotic affections of the lungs

*Sterigmatocystis nidulans* Eidam 1883

**Synonym**—*Aspergillus nidulans* Eidam 1883

Mycelium of a greenish colour Conidiophores are erect 0.5 to  
0.8 millimetre in length Presence of primary sterigmata sup-

**Pathogenicity** — This *Aspergillus* was found by Lindt in a case of otomycosis, according to this author, it is very pathogenic for rabbits

*Aspergillus herbariorum* Wiggers, 1780

**Synonyms** — *Uucor herbariorum* Wiggers 1780 *Aspergillus*

This  
which

Florida

*Aspergillus repens* De Bary 1870

1166 / 1000 p  
mann in the ear  
pathogenic rôle is doubtful

1166

*Aspergillus pictor* R Blanchard  
1895

**Synonym** — *Trichophyton pictor*  
R Blanchard 1895

The term *Aspergillus* (*Trichophyton*) *pictor*, introduced by Blanchard in 1895, before the plurality of species of the fungi found in punta was demonstrated is now used to denote the species of *Aspergillus* which is found in the pure violet variety of punta



FIG 534 — *Aspergillus pictor*  
R BLANCHARD  
(After Montoya y Florez)

This fungus shows the typical growth of *Aspergillus*. It grows easily on agar the growth has at first a violet color which changes into greenish to yellow at a later period. The growth is very luxuriant on the medium on which it is grown comparatively

with certainty



cause any disturbance. The second causes various inflammatory symptoms.

5 ASPERGILLOSIS OF WOUNDS AND ULCERS—In wounds not properly treated and in old ulcers *Aspergillus* have been occasionally found.

6 ASPERGILLOSIS OF THE URETHRA—In several cases a black urethral discharge has been observed due to *Sterigmatocystis nigra*.

7 ASPERGILLOSIS OF THE SKIN—Montoya's investigation has shown that several varieties of *Aspergillus*. Another important variety was believed until recently to be *A. tohelaui* Wehmer 1903. In *A. imbricata* never show aspergillar fructifications and cannot be considered to be *Aspergillus*.

*Aspergillus* may also cause some varieties of mycetoma.

## PYRENOAMYCETES

### CLASS BASIDIOMYCETES

Basidiomycetes have a septate mycelium and are devoid of sexual reproduction. They reproduce by formation of basidia. Other accessory fructifications may be present—as for example chlamydospores. The basidia are of two principal types (1) auto-basidia (2) protobasidia.

lateral inserted sterigma

B Septated by walls intersecting at right angles each cell ending in an elongated tubular sterigma.

Of the Basidiomycetes one species (*Ustilago phoddytes*) of the family Ustilaginæ is of special importance while two others (*U. carbo* and *Tilletia lutea*) may produce otomycosis.

#### *Ustilago hypodytes* Schlecht

Synonyms.—*Dendrodichium microsporus* Brigg. *Sporotrichum dermatodes* Kane

g rise at their  
s a spore  
chief forms —  
spore from a



FIG 535—*STERIGMATOCYSTIS*  
FRUCTIFICATION  
(After Brumpt)



FIG 536—*Sterigmatocystis*  
*nidulans* EIDAM  
(After Eidam from Brumpt)

in certain nests, hence the name *S. nidulans*. In man it has been found in several cases of otomycosis. Nicolle and Pinoy have found it or a very similar species in a case of mycetoma with white granules.

#### General Remarks on Mycoses due to Species of the Family Aspergillaceae.

These mycoses are generally called aspergillomycoses. They have been recorded several times in man.

1 ASPERGILLOSIS OF THE LUNGS. ASPERGILLAR PSEUDO TUBERCULOSIS, PNEUMOMYCOSIS OF ASPERGILLAR ORIGIN.—*Aspergilli* develop sometimes in the mucosa of the trachea of the bronchi, and even in the pulmonary alveoli, without giving rise to any pathogenic effect. In other cases the fungi induce pseudo-membranous and ulcerative lesions. A very serious affection is a form of pseudo-tuberculosis (pseudo-tuberculosis aspergillina), characterized by the presence of mycotic nodules in the lungs, liver, kidneys and other organs. This affection due to *A. fumigatus* is very common in

for feeding the pigeons.

2 ASPERGILLOSIS OF THE EYE.—*A. fumigatus* has been found several times in ulcers of the cornea (keratomyces aspergillina).

3 ASPERGILLOSIS OF THE EAR (OTOMYCOSIS ASPERGILLINA).—

in some  
nests  
g with

*ngatus*

have been found in the nasal cavities, the first apparently does not

## PLATE VI

### NOCARDIAS AND ASSOCIATED ORGANISMS

1. Hair, natural size *Trichomyces flava*
2. *Trichomyces flava*.
3. *Trichomyces flava*.
4. *Trichomyces nigra*
5. *Trichomyces rubra*.
6. Hair, natural size *Trichomyces rubra*
7. *Rhodococcus castellani* (Chalmers and O'Farrell, 1913)
8. *Nocardia indica* (Kanthack, 1893)
9. *Nocardia convoluta* Chalmers and Christopherson, 1916
10. Hair, natural size *Trichomyces nigra*
11. *Cohnistreptothrix tenuis* (Castellani, 1911), branching.
12. *Cohnistreptothrix tenuis* (Castellani, 1911), bacillary forms.
13. *Cohnistreptothrix tenuis* (Castellani, 1911), long form
14. *Cohnistreptothrix tenuis* (Castellani, 1911), branching forms and coccid forms from a culture

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tc  
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ng  
100  
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Trichophyton and th  
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*Phyton rubrum*

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Bruxelles  
Soc de Path Etot  
gricultural Research Octobre

PLATE VI.





- A Accessory fructifications present in the form of closed or open receptacles—Subclass 1 *Deuteromycetes* Saccardo 1886 *emendat* Vuillem 1910
- B No such accessory fructifications present. Reproduction by means of spores isolated or in groups situate on isolated or fasciculated hyphæ—Subclass 2 *Hyphales* Vuillemin 1910

SUBCLASS 1 DEUTEROMYCETES SACCARDO 1886 *emendat* VUILLEMIN 1910

**Definition**—Fungi Imperfecti possessing accessory fructifications in the form of open or closed receptacles

- A Conidiophores minute and enclosed in a perithecium—Order 1

B

d on a disc  
2 *Vullem*

SUBCLASS 2 HYPHALES VUILLEMIN 1910

**Synonyms**—*Nematomyces* Nees 1816 *Hyphomycetes* Martius 1817 *pro parte* *Hyphomycetes* Fries 1833 *Moniliales* Clements 1909

**Definition**—Fungi Imperfecti with hyphæ more or less developed lax or more or less compact superficial or subsuperficial or more rarely as in man vertebrates and insects endoparasitic Conidiophores never situate in closed or on open receptacles. Reproduction by means of spores isolated or in groups situate on isolated or fasciculated hyphæ

**Remarks**—The *Hyphales* of Vuillemin correspond to the *Ues* 18 50 of name

Vuillemin's classification appears to us to be more suitable from the point of view of tropical medicine and therefore we adopt it in this chapter but in order to permit comparison we give the outlines of the old division of the *Hyphomycetes* which is as follows—

ORDER HYPHOMYCETES Fries 1833

- A *Hyphomycetes* with hyaline or brightly coloured hyphæ which do not cohere in fascicles and with concolorous conidia—Family 1 *Mucidinaceæ* Lmk 1809
- B *Hyphomycetes* with dark coloured or black hyphæ rarely hyaline and then with dark coloured conidia. The hyphæ do not cohere into fascicles—Family 2 *Dematiaceæ* Fries 1832
- C *Hyphomycetes* with hyaline or dark coloured hyphæ which do not cohere into fascicles—Family 3 *Uromyces* Fries 1832
- D *Hyphomycetes* with hyaline or dark coloured hyphæ compacted into a globose discoid or verruciform body called a sporodochium—Family 4 *Tuberculariaceæ* Ehrenberg 1818

## CHAPTER XXXIV

### FUNGI IMPERFECTI

Preliminary—Fungi imperfecti—Hyphales—Vuillemin's classification—Microsiphonales—Thallosporales—Hemisporales—Conidiosporales—References

#### PRELIMINARY

We now come to the last class of Schroeter's Eumycetes—viz the Fungi Imperfecti that is to say Eumycetes with a septate mycelium and with spores which are not contained in asci or basidia but are carried on conidiospores which may or may not be enclosed in pycnidia

This class contains a large number of genera of importance in tropical medicine but everyone who has studied these fungi must

new host

n which

prevents the same fungus being variously classified in different stages of its life history We adopt it for purposes of utility

#### CLASS FUNGI IMPERFECTI Fockel 1869

Synonym—*Deuteromycetaceæ* Saccardo (Sylloge vol xvi p 825)

Definition.—Fungi almost invariably minute in which asexual reproduction takes place by means of conidia produced on conidiophores which are either enclosed in perithecia placed on discs or unprotected

Remarks



conidium forms secondary functional spores called Deuteroconidia which are the reproductive spores

III The *Conidia* (sing *Conidium*) are spores which differ from the thallus in being incapable of forming new spores or hyphae while still attached to the parent mycelium They show great

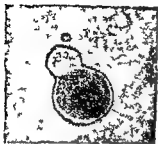


FIG 337—A BLASTOSPORE FROM *Cryptococcus myrmecina* CHALMERS AND CHRISTOPHERSON



FIG 338—A CHLAMYDOSPORE FROM *Trichophyton curvis* CHALMERS AND MARSHALL.

variety of form being rounded stellate simple or septate (staurospore), needle-shaped simple or septate (scolecospore) or spirally twisted simple or septate (heliospore) and of structure being simple (amerospore) or divided by septa (didymospores with two cells and one transverse septum phragmospores with two or more



FIG 539—A HEMI SPORE DEVELOPMENT FROM *Hems spora stellata* VUILLEMIN

(a) Protoconidia  
(b) Deuteroconidia



FIG 540—A HEMI SPORE FROM *Hems spora stellata* VUILLEMIN



FIG 541—ALBURIOSPORES FROM *Aleurisma flavissimum* (LINK, 1816) EMENDAVIT CHEVALIER 1836

(After Vuillemin)

transverse septa and three or many cells dictyospores in which there are longitudinal septa as well as transverse septa) The variation of colour is also of importance as a means of classification The colouring matter usually occurs in the membrane

Their number is also important as they may be single formed in

With reference to the name of Family 3, this has been changed from *Stilbaceæ* Fries, 1825 to its present name, because Juel has demonstrated that some of the species of the genus *Stilbum* belong

species of fungi and this is based upon the colour of the hyphæ and conidia and the number of septa in the spore, as will be indicated when detailing the hyphales. Clements has published a most useful key in English to Saccardo's 'Sylloge'.

Leaving this form of classification we will consider Vuillemin's new system.

**Vuillemin's Classification.**—Vuillemin has pointed out a difficulty which we have also experienced in classifying the fungi of

the 'spore' in order to form the order of the class. Hemphill has

duction. The various forms of the *Thallospore* are named *Blastospores*, *Arthrospores* and *Chlamyospores*.

A . . . . .

*Diaspore* as in *Cryptococcus* or may be an elongated filament.

An *arthrospore* is a *thallospore* developed by the disarticulation of the thallus which has thick walls. The *chlamyospore* is a *thallospore* loaded with food material.

If the *Hemispore* (Figs 539 and 540) starts by a differentiation from the thallus, the 'Protoconidium,' but this remains where formed while the hypha continues to grow. Eventually the prot-



basipetal chains which are more or less persistent or they may be glued together into masses by agglutinating material

The Conidia show two main types—viz the Aleutospore and the Conidium Verum

An Aleutospore (Fig 541) is not a true conidium. It may be terminal, lateral or intercalary but in each case it is not originally distinct from the thallus and is only set free by the death of the filament to which it is attached. It has the morphological significance of a chlamyospore.



FIG 542—SPOROPHORES AND CONIDIA FROM A CLADOSPORIUM



FIG 543—A PHIALIDE FROM *Spicaria ophiodii* VUILLEMIN (a) PHIALIDE (After Vuillemin)



FIG 544—A PROPHIALIDE FROM *Urophiala amycephala* VUILLEMIN 1910 (a) PROPHIALIDE (After Vuillemin)

A Conidium Verum (Fig 542) is quite distinct from the thallus from which it is easily detachable. It may or may not be carried on a conidiophore, the varieties of which are as follows—

A Sporophore

A Ph

and interposed between the sporophore and the conidia

A Prophialide (Fig 544) is a special article on the sporophore from which phialides arise

Having thus made clear Vuillemin's differentiation of the spores it is possible to consider his classification of the Hyphales

Classification—Vuillemin divides the Hyphales as follows—

A Mycelium composed of fine bacilliform hyphae in which the nuclei are usually indistinct—Order 1 *Microspinales* Vuillemin 1912

B Mycelium not so composed—

1 Reproduction by thallospores—Order 2 *Thallosporales* Vuillemin 1910

2 Reproduction by hemispores—Order 3 *Hemisporales* Vuillemin 1910

3 Reproduction by conidia—Order 4 *Conidiosporales* Vuillemin 1910

## ORDER I MICROSIPHONALES Vuillemin 1912

man animals and plants or saprophytic

**Remarks**—This is a most interesting order as its members (Fig 545) are nearly always mistaken at first sight by persons unaccustomed to them for bacilli (Fig 548). Hence the descriptions of bacilli as the cause of so many fungal diseases due to these organisms among which could be classified the diphtheria organisms the tubercular and leprotic bacilli.

Thus one form of division into families is as follows—

- A **Nocardiaceæ** Castellani and Chalmers 1918 **Synonyms**—  
*Actinomyces* Lachner Sandoval 1898 *Trichomyces*  
 Petrusky 1903

**Definition**—Microsiphonales with a mycelium

**Type Genus**—*Nocardia* Tomi and Trevisan 1889

- B **Mycobacteriaceæ** Mico 1909 **Definition**—Microsiphon

nd Neumann

nd Neumann

with the tubercle bacillus as a type

We have however placed the *Mycobacteriaceæ* under the *Streptomyces* and therefore have only the *Nocardiaceæ* to consider

## FAMILY NOCARDIACEÆ

**Synonyms**—*Actinomyces* Lachner Sandoval 1898 *Trichomyces* Petrusky 1903

**Definition**—Microsiphonales with a mycelium

**Type Genus**—*Nocardia* Tomi and Trevisan 1889

**Classification**—Until quite recently all the species of this order were considered to belong to one genus—*Nocardia* Tomi and Trevisan 1889 but Pinoy has made an excellent subdivision separating certain species into another genus which he calls *Cohistanthrix* Pinoy 1911. In doing this he points out that the original discoverers of actinomycosis—viz Harz and Bollinger in 1877 and Rivolta in 1878—thought that they were dealing with one organism but when cultures were attempted it became apparent that more than one organism was implicated. Thus Bostrom isolated a parasite which grew well aerobically producing a dry membrane on the surface of broth and capable of growth at 20° C. on

grew anaerobically and was not capable of growth at ordinary Euro scale—  
 conta  
 into l  
 into  
 This

Wright maintains that this organism is the true cause of actinomycosis and that *N. bovis* is merely a contamination but this cannot be accepted

There are therefore two distinct organisms which can cause

A Grows aerobically easy of cultivation and producing arthrospores (Fig 549)—Genus 1 *Nocardia* Toni and Trevisan 1889

B Grows best anaerobically but can often grow aerobically, difficult of culture and not producing arthrospores—Genus 2 *Cohnistreptothrix* Pinoy 1911

**Genus *Nocardia* Toni and Trevisan 1889**

**Synonyms**—*Actinomyces* Harz 1877 *nec* Meyen 1829 *Discomyces* Rivolta 1878 *nec* *Discomycetaceæ* Fries 1836 *Bacterium* Affanasieff

1897 *nec* Wauters 1833 *Cladothrix* Macé 1897 *nec* Cohn 1875

**Definition**—Nocardiaceæ growing aerobically usually easy of culture and producing arthrospores

**Type Species**—*Nocardia bovis* (Harz 1877)

**Nomenclature**—Bollinger = ray fungus (*Nocardia bovis*) belongs to a genus of which the correct name is *Nocardia* Toni and Trevisan 1889 a term derived from Nocard the celebrated French parasitologist who was the first investigator to clearly recognize this fungus in France We state that it is the correct name for the following reasons—



FIG 545—MICROSIPHONAL S FROM COHNI STREPTOTHRIX TENNIS CASTELLANI 1911

- 1 It is the oldest name against which no objections can be raised
- 2 It has been formally adopted by the Botanical Section of the First International Congress of Pathology
- 3 The objections to the other names in use are as follows —

(a) *Streptothrix* as proposed by Rossi Doria cannot be used as it was originally suggested by Corda in 1826 for *S. f. ssa* which is a totally different fungus

synonyms of the organisms to which they were wrongly applied because of the priority of Corda's name

- (b) *Discomyces* was used by Rivolta in 1878 merely as a trivial name and though it has not been applied to any other genus still the word *Discomycetaceæ* was introduced in 1836 by Fries for a large fungal family and has come into general use and therefore has the double claim of priority and general use and as its type genus should bear the name *Discomyces* confusion is bound to arise if the same term is retained as the generic name of Bollinger's organism
- (c) *Bacterium* was suggested by Affanasieff in 1888 but Ehrenberg had used this name in 1830 for the organisms popularly known as bacteria and therefore Affanasieff's suggestion falls to the ground
- (d) *Oospora* as utilized by Sauvageau and Radais in 1897 is not available because it is younger than the name *Nocardia* and because it was previously used in 1833 by Wallroth for certain fungi previously classified as *Torula* Persoon 1801
- (e) *Cladothrix* as brought forward by Mace in 1897 cannot be used because the name *Nocardia* has priority and because it was originally used by Cohn in 1875 for the organism *Cladothrix dichotoma* which is septate and is only falsely branched and hence is quite different from Bollinger's fungus

**Remarks.**—The genus *Nocardia* contains a large number of species which live saprophytically in soils from whence their spores can be spread by the agency of air or water to sewage sputum etc. Some of them have acquired parasitic habits living in plants in which they cause root tubercles or in other instances tumours with ray fungi thus somewhat resembling the actinomycosis of animals. They have also been found living in molluscs and in the alimentary canals of larval insects as well as in the form of pathogenic fungi in reptilia aves and mammaha in which they mostly occur in the

grew anaerobically and was not capable of growth at ordinary

Wright maintains that this organism is the true cause of actinomycosis and that *N. bovis* is merely a contamination but this cannot be accepted

There are, therefore, two distinct organisms which can cause

- A. Grows aerobically, easy of cultivation, and producing arthrospores (Fig 549)—Genus 1, *Nocardia* Toni and Trevisan, 1889
- B Grows best anaerobically, but can often grow aerobically, difficult of culture, and not producing arthrospores—Genus 2, *Cohniastreptothrix* Pinoy, 1911

Genus *Nocardia* Toni and Trevisan 1889

1892 nec Wallroth, 1833, *Cladothrix* Macé, 1897, nec Cohn, 1875

Definition.—Nocardiaceæ growing aerobically, usually easy of culture, and producing arthrospores

Type Species.—*Nocardia bovis* (Harz, 1877)

(A) wt an

Nocard, the celebrated French parasitologist, who was the first investigator to clearly recognize this fungus in France We state that it is the correct name for the following reasons—



FIG 545—MICROSIPHONALIS FROM COHNI- streptothrix tenuis CASTELLANI 1911



Section II Parasitica Foulerton 1910

The parasitic section can be classified into three subsections as follows —

No	Test	Subsection 1 Majora	Subsection 2 Minora	Subsection 3 Brevis
1	Cultivation at 22° C and 37° C	Easy	Not difficult	Difficult at 37° C Usually nil at 22° C
2	Growth	Spreading	Circumscribed	Slight
3	Efflorescence	Bright chalky	Dull powdery	Usually absent
4	Hyphe branching	Well marked	Poorly marked	Rare hyphae often bacilliform
5	Acid fast species	Rare	Common	Rare
6	Odour of cultures	Earthy or mouldy	Absent or faintly as 1	Sometimes faeculent
7	Liquefaction of gelatine and blood serum	Often present	Rare and usually only one liquefied	Often very slight indications
8	Potato	Growth	Usually growth	Often no growth
9	Diastatic action	Often present	Usually absent	Not known

SUBSECTION I Majora

This subsection contains the following species —

1 N

1895

2 N

Roger Bory and

January 1909

3 *Nocardia modore* (Thury 1897) —  
*Cladonia* —

4 *Nocardia luteola* (Foulerton 1910) —  
*Streptothrix luteola* Foulerton 1910

5 *Nocardia appendicis* Chalmers and Christopherson 1916  
*Streptothrix hominis* III Foulerton 1910  
*Streptothrix hominis* IV Foulerton 1906



FIG 546 — *Nocardia convoluta*  
CHALMERS AND CHRISTOPHERSON  
1916 HYPHA SHOWING BEADING  
AND ALSO COMMENCING SEPARA-  
TION INTO THREE PORTIONS  
(X 1500 DIAMETERS)



FIG 547 — *Nocardia convoluta*  
TO SHOW BRANCHING

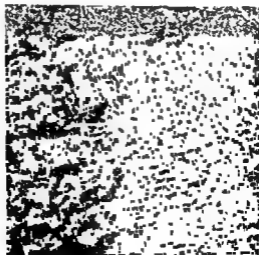
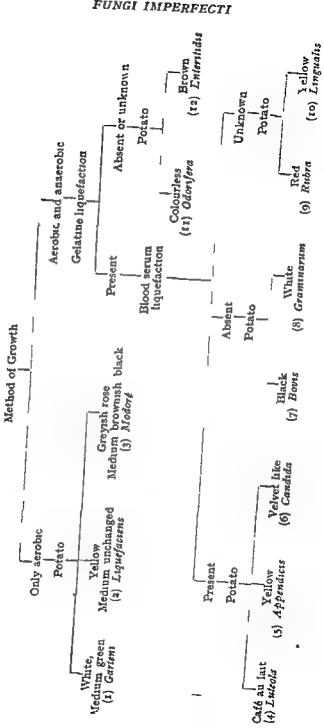


FIG 548 — *Nocardia convoluta* IN SITU IN A GRAIN TO SHOW BACILLIFORM  
APPEARANCE

DIAGNOSTIC TABLE OF THE SPECIES OF THE SECTION *PARASITICA*, SUBSECTION *MAJORA*





- Nec Streptothrix hominis* III Foulerton 1905 = *N. bo*  
*Nec Streptothrix hominis* IV Foulerton 1910 = *N. bo*  
*Nec Streptothrix hominis* III Foulerton 1910  
 12 *Nocardia nigra* (Castellani 1913)  
     Synonym *Streptothrix nigra* Castellani 1913  
 13 *Nocardia pypers* Castellani and Chalmers 1918

As there is so much confusion with regard to the specific name *hominis* Chalmers and Christopherson proposed that—

- These species may be separated as given on p 1055

### SUBSECTION 3 *Brevis*

This subsection contains —

- 1 *Nocardia valvulae* (Luginger 1904) —  
 2  
 3  
 4 *Noc*  
 5 *Noc*  
     Gasperini's *Streptothrix* 1890  
 6 *Nocardia krausei* (Chester 1901) —  
     *Streptothrix krausei* Chester 1901  
 7 *Nocardia foulertoni* Chalmers and Christopherson 1916 —  
     *Streptothrix hominis* Foulerton 1902  
     *Streptothrix hominis* I Foulerton 1906  
 8 *Nocardia londonensis* new name —  
 9  
 10  
     *Streptothrix hominis* Hayo Bruns 1899  
 11 *Nocardia berestneffi* new name  
     *Streptothrix* cases 1 and 2 Berestneff 1897  
 12 *Nocardia equi* (Dean 1900) —  
     *Streptothrix* from a horse of Dean 1900  
     Probably the organism described by Norris and Lar  
     should come here but we have been unable to see  
     description

These species may be differentiated as given on p 1056

*Nocardia madurae* R Blanchard 1895  
*Micrococcus pelletieri* Laveran 1906  
*Oospora pelletieri* Throux and Pelletier 1912  
*Nocardia pelletieri* Pinoy 1912  
*Nocardia riseres* Verdun 1912 (?)

- 4 *Nocardia leishmani* Chalmers and Christopherson 1916 —  
 New acid fast streptothrix pathogenic to man and  
 animals described by Birt and Leishman in 1902

" " " " " " " " " " " "

9 " " " " " " " " " " " "

10

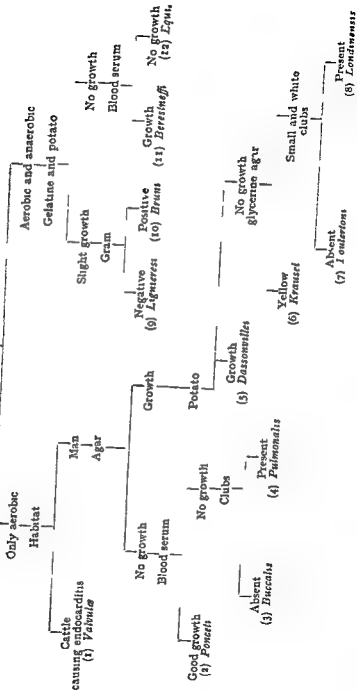
MacCallum by Schabud probably belong to this  
 species ruet by

- 11 *Nocardia hominis* (Berestneff 1897) —

*Nec Streptothrix hominis* Hayo Bruns 1899  
*Nec Streptothrix hominis* Gollerton 1907  
*Nec Streptothrix hominis* II Foulerton 1910

DIAGNOSTIC TABLE OF THE SPECIES OF THE SECTION *PARASITICA* SUBSECTION *BREVIS*

Method of Growth







**Cultures.**—The fungus grows easily on agar, glycerinated agar gelatine, broth, potato and other media. Optimum temperature, 35° to 37° C. On glycerinated agar the fungus grows fairly rapidly, giving rise to small dry, coarsely granular, brown-yellowish colonies, which latter coalesce together. The fungus is Gram-positive, but not acid-fast.

**Saprophytic Life.**—The fungus is found saprophytic on the spikelets of some cereals (*Phleum pratense* Linnæus, *Hordeum murinum* Linnæus etc.)

**Pathogenicity.**—The experimental reproduction of the disease by

*N. bovis sulphurea-alba* *N. bovis luteo rosea*. Caminiti has described a variety which he thinks may be a new species. *N. lanfranchii* Luigi Sani is also a variety of this *Nocardia*.

#### *Nocardia rosenbachii* Kruse, 1896.

**Synonym.**—*Streptothrix rosenbachii* Kruse 1896

Isolated by Rosenbach in a case of dermatitis, called by him 'erysipeloid'. Mycelial threads very slender, some terminate in club-like swellings. Can be cultivated on the usual laboratory media. Does not liquefy gelatine.

#### *Nocardia asteroides* Eppinger, 1890.

**Synonyms.**—*Cladothrix asteroides* Eppinger 1890, *Streptothrix eppingeri* Rossi Doria, 1891, *Oospora asteroides* Sauvageau and Radais 1892, *Nocardia asteroides* R. Blanchard 1895, *Discomyces*

g 1909  
Mycelial  
become  
ungus is  
it grows  
Inocula  
pseudo

tuberculosis histologically indistinguishable from true tuberculosis. McCallum has found a *Nocardia* very similar to *N. asteroides* in a case of peritonitis.

#### *Nocardia indica* Kanthack, 1893.

**Synonyms.**—*Streptothrix madure* H. Vincent, 1894, *Nocardia madure* R. Blanchard 1895, *Micrococcus pelleteri* Laveran, 1906, *Oospora pelleteri* Thiroux and Pelleter 1912, *Nocardia pelleteri* Pinoy, 1912

It causes Vincent's white mycetoma, very commonly found in Africa and Asia. The 'grains' which are found in the pus of such cases are soft, white or slightly yellowish and have a mulberry-like surface. At the periphery of the grains radiating filaments are



**Nocardia pijperi** Castellani and Chalmers 1919

Discovered and described by Pijper in a case of chronic bronchitis in South Africa. The fungus is non motile Gram positive not acid fast. It grows on agar giving rise to small whitish colonies becoming visible after forty eight to seventy two hours. The

guinea pigs by intraperitoneal injection. Nodules develop on the peritoneum consisting of an outer wall of epithelioid cells and leucocytes surrounding a cavity filled with fluid in which asteroid colonies of the fungus are found.

**Nocardia candida** Petruschki 1901

Found in sputum

**Nocardia aurea** Du Bois St Séverin 1902

Found in a case of ulcerative conjunctivitis

**Nocardia odorifera** Rullman and Perutz 1898

Found by Rullmann in a case of chronic bronchitis. Merely a synonym of *N. chromogena* Gasperini

✓ **Nocardia liquefaciens** Hesse 1892

Synonym — *Cladothrix liquefaciens* Hesse 1892

Gives rise to white colonies on gelatine and later liquefies the medium. Was isolated from a case of actinomycosis. Liquefies serum

✓ **Nocardia poncetii** Verdun 1912

Not cultivable on agar or gelatine. Grows well on serum where it takes a bacillary like form. Found by Moorhof Dor and Poncet in a case of myomycosis resembling actinomycosis

**Nocardia fuses** Karwacki 1911

Isolated by Karwacki from the sputum of a tubercular patient. Dark yellowish colour

**Nocardia luteola** Foulerton and Jones

Found in a case of purulent conjunctivitis

**Nocardia carnea** Rossi Doris 1891

Isolated by Baldoni from a case of chronic bronchitis. Red colonies

✓ **Nocardia garteni** Brumpt 1910

Synonyms — *Cladothrix liquefaciens* No 2 Garten 1895. *Discomyces garteni* Brumpt 1910

Liquefies gelatine. On potatoes gives rise to white colonies while the medium takes a greenish colour. Was isolated from a case of actinomycosis

**Nocardia enteritidis** Pottien 1902

Found in cases of enteritis by Pottien

found but claviform swellings are usually absent mycelial threads are always very slender ( $\chi$  to  $1\frac{1}{2} \mu$ ) The fungus can be grown on the ordi — — — — —

forms di  
phery  
grains

short chains of small conidial elements Gram positive but not acid fast

It is to be noted that this fungus very rarely produces bone lesions Most strains are inoculable into monkeys

*Nocardia dassonvillei* Brocq Rousseu 1907

Synonym — *Streptothrix foersteri* (Gasperini 1890)

Very thin ramified mycelial threads which easily become fragmented and dissociated into bacillary like bodies Numerous spherical coccus like bodies (spores) present Gram positive The fungus grows fairly well on gelatine giving rise to small white

su<sub>00</sub> st that the fungus may have been present in the rice-powder as the same fungus is known to be found in several decaying cereals

Gasperini isolated in 1890 from the air a *Nocardia* which he identified as *N foersteri* Further researches (Landrieu) have shown Gasperini's fungus to be *N dassonvillei*

*Nocardia decussata* Langeron and Chevalier 1912

Synonym — *Discomyces decussatus* Langeron and Chevalier 1912

Found by Langeron and Chevalier in a patient presenting peculiar whitish dry squamous patches The fungus grows on

*Nocardia pulmonalis* H Roger Bory and Sartory 1900

ba<sub>00</sub> — — — — —  
bacillary like  
(altose broth)  
some branch  
fine mycelial

threads are very fragile and become fragmented into strings of bacillary or coccus like bodies

Pathogenicity — Causes a type of pseudo tuberculosis In the expectoration occasionally small white granules composed of masses of the fungus are present Sartory has found the fungus in a case of otitis

1896, *Discomyces minutissimus* P Verdun 1907, *Microsporoides minutissimus* Neveu Lemaire 1906 *Oöspora minutissima* Ridet 1911

Mycelial threads extremely thin ( $0.6 \mu$ ) seldom ramified. The mycelial segments get easily dissociated and have then the appearance of bacilli. Is the cause of erythrasma. Michele Ducrey and Reale claim to have cultivated it.

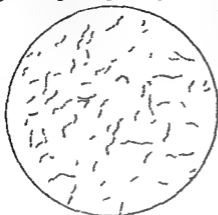


FIG 554 — *Nocardia minutissima* a BURCHARDT



FIG 555 — *Cohniastreptothrix tenuis* CASTELLANI

✓ *Nocardia convoluta* Chalmers and Christopherson 1916

**NOCARDIA** — Gram positive but not acid fast without club formations found parasitic in man easy of cultivation growing aerobically and anaerobically at  $22^{\circ} \text{C}$  and  $37^{\circ} \text{C}$  with a marked preference for alkaline media and with the production of good but limited growths on the different agars and the same at first on blood serum and potato on which however it becomes more profuse later. Not liquefying gelatine but causing liquefaction of inspissated ox blood serum without diastatic action. Colonies usually somewhat translucent when young of a light to warm buff colour (Ridgway ■ Plate VV 17 O V f or d) and either convoluted or having the appearance of a jelly turned out of a mould later developing a whitish powdery efflorescence without distinct odour never pigmenting the medium on which it is grown not fermenting or peptonizing milk. Non pathogenic for monkeys and other laboratory animals.

**Remarks** — It is fairly frequently found in the actinomycotic type of Madura foot in Khartoum, Anglo Egyptian Sudan.

*Nocardia nigra* Castellani 1913

*Nocardia* Gram positive some strains acid fast no definite club formations. Grows aerobically and anaerobically at  $22^{\circ} \text{C}$  and  $35^{\circ} \text{C}$ . Colonies on maltose agar and ordinary agar are black. Most strains liquefy gelatine.

*Nocardia lutea* was found by Christopherson and Archibald in 1918 in the lachrymal canal of a case in Khartoum.

*Nocardia buccalis* H Roger Bory and Sartory 1909

Synonyms — *Oospora buccalis* H Roger Bory and Sartory 1909

> 8  $\mu$ ) Some ramified mycelial  
 1g fragmented into strings of  
 Can be cultivated



FIG 552 — *Nocardia pulmonalis*  
 H ROGER BORY AND SARTORY  
 (After Roger and Sartory)



FIG 553 — *Nocardia buccalis*  
 ROGER BORY AND SARTORY

Pathogenicity — May give rise to a form of stomatitis somewhat resembling thrush. It has been found also in tonsillar abscesses

*Nocardia lasserrei* Verdun 1912

situated on  
 thin (0.5 to  
 1) cultivated  
 intracerebral

injection

*Nocardia linguæ* Guiguen 1908

Synonyms — *Oospora linguæ* Guiguen 1908 *Discomyces linguæ*  
 (Brumpt 1910)

*coccis linguæ pilosa*

*Nocardia rivierei* Verdun 1912

Isolated by Rivière (1895) in a case of multiple abscesses. Is cultivable on ordinary media

*Nocardia appendicis* Chalmers and Christopherson 1916

Synonyms. — *Streptothrix hominis* Foulerton 1906 *Oospora hominis* Ridet 1911

Discovered by Foulerton in a case of multiple abscesses was present also in the expectoration of the same patient. Foulerton has found similar fungi (*N. hominis III*) in cases of appendicitis

*Nocardia minutissima* Burchardt 1869

Synonyms — *Microsporium minutissimum* Burchardt 1869  
*M. gracile* Balzer 1893 *Sporotrichum minutissimum* Saccardo

*polymorphus* Berestneff 1898 may be the same as the chromogenic anaerobic streptothrix obtained from human pus by Neschezadimenko in 1908

**Classification** —The species included in this genus are —

- 1 *Cohnstreptothrix silberschmidti* Chalmers and Christopherson 1916 —

*logie* xxvii and further cases in *Zeitschrift für Hygiene* (1901) xxxvii

- 2 *Cohnstreptothrix cuniculi* (Schmorl 1891) —  
*Streptothrix cuniculi* Schmorl 1891

? .. .. .

?

?

- 3 *Cohnstreptothrix nesche adimenki* Chalmers and Christopherson 1916 —

This name is given to distinguish the obligatory anaerobic streptothrix found by Neschezadimenko in 1908 in human pus and described in the *Centralblatt für Bakteriologie* xlv1

? *Coccobacillus pseudo actinomycosis polymorphus* Berestneff 1898

- 4 *Cohnstreptothrix americana* Chalmers and Christopherson 1916 —

This name is given to distinguish the streptothrix which only grows under partial anaerobic and aerobic conditions obtained from a liver abscess by Blodmfield and Bayne-Jones in 1915 and described in *Johns Hopkins Hospital Bulletin* xxvi No 792

- 5 *Cohnstreptothrix israeli* (Kruse 1896) —

*Streptothrix israeli* Kruse 1896

*Streptothrix spitzii* Lignieres 1903

Possibly the streptothrices described by Doyen in 1891

dt

3

- 6 *Cohnstreptothrix thibiergei* (Ravaut and Pinoy 1909) —  
*Discomyces thibiergei* Ravaut and Pinoy 1909

## Genus Cohnistreptothrix Pinoy, 1911

**Definition**—Nocardiaceæ growing best anaerobically but can grow aerobically, usually difficult to cultivate and do not produce arthrospores

**Type Species**—*Cohnistreptothrix israeli* (Kruse 1896)

**Historical**—In 1891 Wolff and Israel published a beautifully illustrated account of a streptothrix which they had isolated from

to make a new species for it under the name *Streptothrix israeli*

genus streptothrix calling the fungus in question *Streptothrix foerstersi* Cohn, 1875 which may be the same organism as *S. auratus* Du Bois de Saint Severin 1895 and must be closely related to *Nocardia tenuis* Castellani 1911 which belongs to the same genus and as its colonies on agar are cerebriform it may possibly be the same as or related to *Streptothrix radiatus* and *S. cerebriformis*

been given by Corda in  
 ingus which is known as  
 of any importance on systemic mycology Therefore as strepto-  
 thrix is not available after many changes the generic name has  
 become *Cohnistreptothrix* Pinoy, 1911 and to this genus *israeli*'s  
 human organism belongs It differs from Bollinger's type of fungus  
 in growing best anaerobically in being difficult to cultivate and in  
 not producing arthrospores Other allied organisms are *Cohni-*  
*streptothrix* etc.

ab-cess in America by Bloomfield and Bowen Jones (1915) Perhaps



- 7 *Cohnistreptothrix foersteri* (Cohn 1874) —  
*Streptothrix foersteri* Cohn 1874  
*Leptothrix oculorum* Sorokin 188x

The aerobic streptothrix of Silberschmidt obtained from a case of dacryocystitis 1901

? *Streptothrix radia'a* Namyslowski 1909

? *Streptothrix cerebriformis* Namyslowski 1909

- 8 *Cohnistreptothrix tenuis* (Castellani 1911) —  
*Nocardia tenuis* Castellani 1911

- 9 *Cohnistreptothrix carougeana* (Gougerot 1909) —  
*Discomyces carougeana* Gougerot 1909  
*Nocardia carougeana* Castellani and Chalmers 1913

These species may be differentiated as given on p 1065

#### *Cohnistreptothrix israeli* Kruse 1896

**Synonyms** — *Streptothrix israeli* Kruse 1896 *Cohnistreptothrix israeli* Pinoy 1911

Found in some cases of human and bovine actinomycosis. It differs from *N. bovis* by being strictly anaerobic. Inoculations of pure cultures have reproduced actinomycotic lesions while so far such experimental lesions have not been obtained by using culture of *N. bovis*.

Wright states that *N. israeli* is the real cause of actinomycosis while *N. bovis* would be only a contamination or a saprophytic agent. We agree however with Pinoy's opinion that the clinical

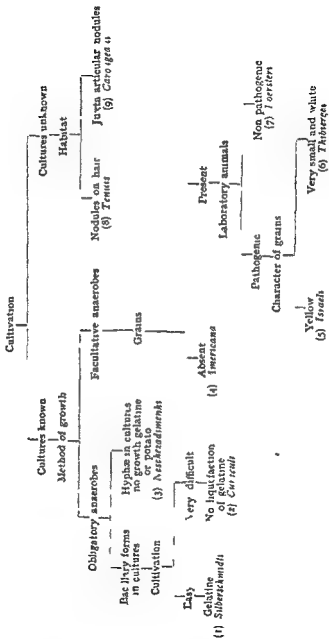
gits in man due to this bacillus

#### *Cohnistreptothrix thibiergei* Pinoy and Ravaut 1909

**Synonym** — *Nocardia thibiergei* Pinoy and Ravaut 1909

Discovered by Pinoy and Ravaut in a case of peculiar nodular affection of the subcutaneous tissues and muscles. In the lesion

DIAGNOSTIC TABLE OF THE SPECIES WHICH MAY POSSIBLY BELONG TO THE GENUS  
COHNISTREPTOTHRIX PINOY 1911



- 7 *Cohnistreptothrix foersteri* (Cohn 1874) —  
*C. foersteri* Cohn 1874

895

*Streptothrix foersteri* Kruse 1896

The aerobic streptothrix of Silberschmidt obtained from  
 a case of dacryocystitis 1901

? *Streptothrix radiata* Namyslowski 1909

? *Streptothrix cerebriformis* Namyslowski 1909

- 8 *Cohnistreptothrix tenuis* (Castellani 1911) —

*Nocardia tenuis* Castellani 1911

- 9 *Cohnistreptothrix carougeauxi* (Gougerot 1909) —

*Discomyces carougeauxi* Gougerot 1909

*Nocardia carougeauxi* Castellani and Chalmers 1913

These species may be differentiated as given on p 1065

#### *Cohnistreptothrix israeli* Kruse 1896

**Synonyms** — *Streptothrix israeli* Kruse 1896 *Cohnistreptothrix israeli* Pinoy 1911

Found in some cases of human  
 differs from *N. bovis* by being  
 pure cultures have reproduced

such experimental lesions have not been obtained by using cultures  
 of *N. bovis*

Wright states that *N. israeli* is the real cause of actinomycosis  
 while *N. bovis* would be only a contamination or a saprophytic  
 agent We agree however with Pinoy's opinion that the clinical  
 features of actinomycosis may be due to several germs in man Pinoy  
 has found *N. bovis* and *N. israeli* in oxen *N. israeli* in most cases  
 but also *N. bovis* the actinobacillus and mixed infections

actinomycosis in man due to this bacillus

*Cohnistreptothrix thibergei* Pinoy and Ravaut 1909

nodular  
 lesions  
 associated  
 with pharyngitis

The fungus is easily cultivated aerobically and anaerobically. On maltose agar it produces small white colonies; on broth it develops only at the bottom of the tube.

*Cohnistreptothrix foersteri* Cohn 1874

**Synonyms**—*Streptothrix foersteri* Cohn 1874; *Oospora foersteri* Radais et Sauvageau 1897; *Discomyces foersteri* Blanchard 1895

Investigation of the fungus which according to him shows a slow growth and gives rise on maltose agar to small cerebriform colonies of a grey stone colour

*Cohnistreptothrix tenuis* (Castellani 1911)

**Synonym**—*Nocardia tenuis* Castellani 1910

Found by Castellani in a nodular affection of the hair of the axillary regions. In the parasitic stage the germ appears in the shape of bacillary like bodies

in length (2 to 10  $\mu$ ) packet

amorphous cementing substr

straight or bent seldom bran

fast. Masses of this fungus embedded in amorphous cementing substance form the nodules of trichomyces flava of the axillary regions. (See I late VI p 103f)

In the N. by p. a. = f. l. =

The red pigment producing coccus on the other hand grows better and shows more pigmentation on ordinary agar than on maltose or glucose agar

### ADDENDUM

A few words may be inserted on certain filamentous vegetal organisms on the classification of which there is much discussion—viz organisms of the genus *Leptothrix* of the genus *Cladothrix* of the genus *Vibriothrix*

#### Genus *Leptothrix* Kutzing 1813

**Definition**—Filamentous fungi with long very thin mycelial threads with no capsule or only a very delicate one non branching non septate generally non cultivable

**Type Species**—*Leptothrix maxima* Miller

The following species concern us —

#### *Leptothrix maxima* Miller 1832

**Synonym**—*L. buccalis maxima* Miller

Long thin filaments unsegmented or with very long segments When treated with iodine and dilute sulphuric acid gives a blue granulo-se reaction Has not been cultivated

#### *Leptothrix innominata* Miller 1882

Morphologically identical with *L. maxima* but when treated with iodine and dilute sulphuric acid does not give a blue reaction Has not been cultivated

#### *Leptothrix racemosa* Miller 1882

Filaments somewhat thicker than those found in the two preceding species On staining shows a peculiar beaded appearance Has not been cultivated

#### *Leptothrix placoides* Dobrzynski

Very long thin filaments Gram positive non motile Gelatine liquefied Growth on agar very slow produces very hard granular colonies Isolated from human mouth by Dobrzynski

#### *Leptothrix filiformis* Flexner 1896

**Synonym**—*Bacillus (Leptothrix ?) pyogenes filiformis* Flexner 1896 Isolated by Flexner from a rabbit Is non motile of difficult cultivation pathogenic

#### *Leptothrix vaginalis* Donné 1885

Found in vagina of women and mammals

#### Genus *Cladothrix* Cohn 1875

**Definition**—Filamentous fungi with mycelial threads very long thin showing pseudo branching The only species concerning us is *Cladothrix dichotoma* Cohn

#### *Cladothrix dichotoma* Cohn 1875

They are  
e of pseudo  
itory media  
layer very  
The organ  
ilar species

#### Genus *Vibriothrix* Castellani 1917

The mycelial articles are motile of very different shape bacillary vibrio like spirillum like at times club-ended Globular or pear shaped bodies

of very variable size may be present Gram negative not acid fast Cultiv

show a predominance of vibriose of (BULLIUM) TO THE (BULLIUM) (BULLIUM) (BULLIUM)

## ORDER II THALLOSPORALES Vuillemin 1910

Definition — This order is characterized by the presence of hyphae resembling those of the Zygomycetes, and by the formation of thallospores. Parasitic on man, animals and plants or saprophytic.

Classification — This order may be divided as follows —

Reproduction by means of the form of thallospore called blastospore—Suborder 1 *Blastosporineæ* Vuillemin 1911.

Reproduction by means of the form of thallospore called arthrospore—Suborder 2 *Arthrosporineæ* Vuillemin 1911.

### SUBORDER 1 BLASTOSPORINEÆ VUILLEMIN 1911

species then it at once became a saccharomyces. The researches of many observers but particularly Busse, tend to show that the

genus *Cryptococcus* Kützing 1833 is good and therefore should find a suitable place in a fungal classification

*Saccharomyces* and its allies and *Cryptococcus* and its allies however so closely related that it is necessary to give some simple scheme whereby laboratory workers and clinicians may easily differentiate those found in pathological work and such a scheme is as follows —

- I In cultures budding forms present mycelium absent only traces thereof present asci present—*Saccharomyces*
- II As I but no asci present—*Cryptococcus*
- III Budding forms present mycelium well developed septate or not branched or not asci present—*Uromyces*
- IV As III but asci not present—*Monilia*
- V Budding forms absent mycelium well developed septate oval or rectangular arthrospores (thallospores) present—*Oidium*

**Classification** : The various families of the Blastosporineæ which we are concerned may be recognized as follows —

- A *Hyphe* not manifestly different from the spores —
  - I Spores not in chains : Usually do not ferment carbohydrates with the production of gas—Family 1 *Cryptococcaceæ* Kützing 1833
  - II Spores in chains : Usually ferment carbohydrates with the production of gas—Family 2 *Oosporaceæ* Saccardo 1880
- B *Hyphe* manifestly different from the spores —
  - I Spores not in chains but arranged verticillately—Family 3 *Enantiothamnaceæ* Chalmers and Arnold 1915
  - II Spores in chains—Family 4 *Haplographiaceæ* Saccardo 1886
  - III Spores in short chains or solitary—Family 5 *Cladosporiaceæ* Saccardo 1886

#### FAMILY I CRYPTOCCACCÆ Kützing 1833

DEFINITION

ascomyces hemi asci proto asci and gymnascales This or is looked upon as a true ascocete and the true ascocete is believed that true ascocetes had the same characters as the ascocete

of very variable size may be present. Gram negative not acid fast. Cultiv

..

## ORDER II THALLOSPORALES Vuillemin 1910

**Definition**—Hyphae with the mycelium composed of hyphae more than one micron in diameter and either short and resembling the conidia or longer and distinct therefrom. Reproduction by means of thallospores. Parasitic on man, animal and plants or saprophytic.

**Classification**—This order may be divided as follows—

Reproduction by means of the form of thallospore called blastospore—Suborder 1 *Blastosporineæ* Vuillemin 1911

Reproduction by means of the form of thallospore called arthrospore—Suborder 2 *Arthrosporineæ* Vuillemin 1911

### SUBORDER 1 BLASTOSPORINEÆ VUILLEMIN 1911

**Definition**—

of many observers but particularly Busse, tend to show that the



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by ~~some~~ but it was ~~Dusse~~ who first proved that they were the true cause of the disease and showed that they were pathogenic to animals. These yeast like organisms were found at the autopsy some thirteen months later to occur in sarcomatous like growths consisting of

by reproduction by budding and no endospores or mycelium were ever seen. It was this that induced ~~Vuillemin~~ to give it the name

as at present is referred to the genus *Cryptococcus* e.g. —  
*Cryptococcus breweri* Verduyn 1912 described in an abscess of the

d  
f

The following have been found in the mouth or throat — *C. sulfureus* Beauverie and Lesieur 1912, *C. lesieurii* Beauverie and Lesieur, 1912, *C. salmonensis* Sartory 1911, *C. guillermondi* Beauverie

**Classification**—The family contains the following genera — *Torula* Persoon 1801, *Cryptococcus* Kützing 1833, *Pityrosporum* Sabouraud 1893, and *Mycoderma* Persoon 1822 which may be differentiated as follows —

A Vegetative elements not elongate —

I *Non-pathogenic* —

II *Pathogenic* —

(a) Vegetative elements with well developed double contour no large globuli of fat Budding takes place with a single bud at a time No thick pellicle on fluid sugar media Cultivated—*Cryptococcus*

(b) Vegetative elements often without double contour Not cultivated—*Pityrosporum*

B Vegetative elements elongate —

In fluid sugar media a thick pellicle containing bubbles of gas is quickly produced—*Mycoderma*

The genera with which we are concerned are *Cryptococcus* and *Pityrosporum*



FIG 556 — DIAGRAM SHOWING  
BUDDING CHARACTERISTIC OF  
*mycoderma* (a) and *Torula* (b)  
(After Hansen)



FIG 557 — *Cryptococcus*  
*myrmeciae*

**Genus *Cryptococcus* Kützing 1833**

Γ α-ω - α - α

Found in abscesses in a woman by Busse. In the pus the fungus presented itself in the shape of oval bodies, with a membrane having a double contour. These elements were arranged in groups, each group embedded in an amorphous substance and surrounded by a capsule. Culturally the fungus showed only roundish budding forms, no mycelium, no asci. Growth on solid media generally white. Gelatine not liquefied. Glucose fermented. Pathogenic to rabbits, white mice, and dogs.

*Cryptococcus lingua-pilosa* Lucet, 1901

Synonym.—*Saccharomyces lingua-pilosa* Lucet, 1901



Attempts to reproduce the disease have failed. Guégen and Thôn believe that the fungus becomes pathogenic when

FIG. 597.—*Cryptococcus lingua-pilosa* LUCET  
(After Lucet from Brumpt)

*Cryptococcus plimмери* Constantin, 1901

Found by Plimmer in many cases of

Cultivation has succeeded only on one occasion when it was found that it produced a white growth on agar and other media. Action on gelatine and other sugars have not been investigated. *Cryptococcus plimмери*, according to some authors, has been applied to cellular changes.

*Cryptococcus degenerans* Vuillemin, 1896

Synonym.  
Found by  
cinomata  
any other  
scarcely kn  
about other

*Blastomyces degenerans* Vuillemin, 1896  
D. = 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50, 51, 52, 53, 54, 55, 56, 57, 58, 59, 60, 61, 62, 63, 64, 65, 66, 67, 68, 69, 70, 71, 72, 73, 74, 75, 76, 77, 78, 79, 80, 81, 82, 83, 84, 85, 86, 87, 88, 89, 90, 91, 92, 93, 94, 95, 96, 97, 98, 99, 100

*Cryptococcus corselli* Neveu Lemaire, 1903

Found by Corselli and Frisco in a sarcoma of the mesenteric glands. The fungus appeared in the tumours under the shape of black masses. It was easily cultivated and found pathogenic to guinea pigs, rabbits, and dogs. Sugar reactions unknown.

*Cryptococcus hessleri* Rettger, 1904

Synonym.—*Blastomyces hessleri* Rettger, 1904

Isolated by Hessler from a small tumour which developed on a patient after a razor-cut. Easily cultivated, especially on alkaline media. Pathogenic for the rabbit and guinea pig. Sugar reactions unknown.

and Lesieur 1912 *C. rogersi* Sartory 1911, and *C. lingua filosa* Lucet 1901

According to most authors *Histoplasma capsulatum* Darling 1906 is not an animal but a vegetal parasite and should be classified as a cryptococcus

The parasite found in cases of chronic ulcerative dermatitis in

American soldiers in the Philippines (Gilchrist and Stokes 1898) is a yeast-like fungus which was first described by Stokes in 1822, and later by Gilchrist and Stokes in 1898.

In 1914 they have named it *C. myrmecia* Chalmers and Christopherson 1914 but they were unable to prove conclusively that it was the ætiological factor though they brought forward some facts to support such a contention

### *Cryptococcus dermatitidis* Gilchrist and Stokes 1898

— — — — — es 1898  
Beurmann

ulcerative dermatitis and later in a case which had been diagnosed as a

tuberculide of the skin After Gilchrist and Stokes's cases other cases of blastomycosis due to an identical or similar organism have been described by Hyde, Oppenheim, Ricketts and others Ricketts considered these fungi to be species of *Oidium* and proposed the name *oidiomycosis* to indicate the disease produced by them

*C. dermatitidis* in the affected tissues has the appearance of a typical yeast—the large globular cells 10 to 16  $\mu$  in diameter reproducing by budding In cultures which are white besides these globular elements rudimentary mycelial tubes may occasionally be found presenting lateral or terminal conidia, asci are absent The fungus does not ferment sugars and there is no formation of a pellicle Gelatine not liquefied



FIG. 558. *Cryptococcus dermatitidis* GILCHRIST AND STOKES (After Gilchrist)

### *Cryptococcus hominis* Vuillemin 1901

Synonyms.—*Saccharomyces* (sp. ?) Baer 1894 *Atelosaccharomyces busse buschki* de Beurmann and Gougerot 1899 *Atelosaccharomyces rudis* de Beurmann and Gougerot 1911

**Cryptococcus niger** Vuillemin

Found by Maffucci and Surleo in 1895 in a pulmonary myxoma of a guinea pig. Cells *in situ* round or ovoid with thick mucilaginous membrane. White on most media on potato brown or black. Gelatine not liquefied. Said to ferment maltose. Nothing known about other sugars.

**Cryptococcus lithogenes** San Felice

**Synonym.**—*Saccharomyces lithogenes* San Felice

Found by San Felice in the lymphatic glands of an ox. Roundish cells with a membrane which is at times calcified. Growths whitish on most media dark brownish after a time on potato. Gelatine not liquefied.

**Cryptococcus granulomatogenes** (San Felice)

**Synonym.**—*Saccharomyces granulomatogenes* San Felice

Isolated from the lung of a hog by San Felice. It grows easily on ordinary laboratory media producing white colonies. It does not liquefy gelatine but it produces slight rose-red pigment on slices of pear and on honey.

**Cryptococcus farciminosus** (Rivolta and Micellone 1883)

"

**Remarks.**—It is often included in hypertrophied endothelial cells and in leucocytes in the lesions in horses suffering from lymphangitis epizootica in Europe Africa Asia and America.

**Cultivation.**—It is of difficult cultivation. Nègre and Boquet have used with success a medium made of agar and dried horse-dung. Sugar reactions are unknown.

**Cryptococcus capsulatus** (Darling 1906)

**Synonym.**—*Histoplasma capsulatum* Darling 1906

**Definition.**—In the affected tissue the parasite appears in round

"

At present there is a consensus of opinion that it is a *Cryptococcus*

**Pathogenicity.**—It is pathogenic for man causing disseminated hyaline pseudogranulomata in the lungs splenomegaly necrotic areas in the liver and ulceration of the small and large intestines.

**Cryptococcus ruber** Demme 1889

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red

ar

**Cryptococcus breweri** Verdun 1912

Synonym *Atelosaccharomyces breweri* Verdun 1912

Found by Brewer and Wood in an abscess of the vertebral column

**Cryptococcus tonkini** Legendre 1911

Synonym — *Blastomyces tonkinsi* Legendre 1911

Found by Legendre in two cases of blastomycosis in Indo China. Tinoy considers that the characters given are not sufficient to create a new species for it

**Cryptococcus sulfureus** Beauverie and Lesieur 1912

Found by Beauverie and Lesieur in certain pharyngeal lesions of a case of typhoid. Ferments slightly dextrose saccharose and lactose

**Cryptococcus lesleuri** Beauverie and Lesieur 1912

Found in a case of stomatitis. Ferments dextrose only

**Cryptococcus salmonis** Sartory 1911

Described by Sartory. Found in human gastric juice. Growth on usual media of a pinkish or reddish colour. Does not ferment any sugar. Slowly coagulates milk

**Cryptococcus guillermondi** Beauverie and Lesieur 1912

Found by Guillermond and Lesieur in cases of stomatitis

Cellular elements *in situ* spherical 10-25 microns surrounded by large mucilaginous capsule. Growth on agar white or slightly yellowish. On potato scanty growth white. Gelatine not liquefied. Does not ferment any sugar

**Cryptococcus harteri** de Beurmann and Gougrot 1913

Synonym — *Atelosaccharomyces harteri* de Beurmann and Gougrot 1913

Cells oval 4-6 and 35 microns. Growth on sugar media and gelatine which is not liquefied white. Does not ferment any sugar. Found by Harter in a case of systemic blastomycosis

**Cryptococcus hudelo** de Beurmann and Gougrot 1914

Cellular elements *in situ* mostly spherical 2-20 microns at times oval easily grown. Colonies white gelatine not liquefied. Growth on potato at first white later yellow and finally reddish or blackish. Found by Hudelo Duval and Loedersch in a case of pericostitis

**Cryptococcus membranogenes** Steinhouse 1916

Cellular elements roundish 7-8 microns in diameter with very distinct double contour. Surrounded by a thick capsule. Easily grown on all the usual culture media. Colonies white. Gelatine not liquefied. Ferments with gas production glucose but not maltose lactose or saccharose. Action on other sugars not known. Very pathogenic for rabbits

Found by Steinhouse in 1916 in a case of scarlet fever showing symptoms of tracheal obstruction

**Cryptococcus epidermidis** Castellani 1914

Found by Castellani in *saccharomyces epidermica*. Cells of very variable size. Has not yet been cultivated

organism with the ætiology of pityriasis simplex capitis and pityriasis alba

Its shape somewhat resembles a budding yeast or a flask. The size varies greatly, the maximum diameter of some individuals may be as much as 10 to 15  $\mu$ , but much smaller forms (3 to 5  $\mu$ ) are found

*Pityrosporum canthel* (Castellani, 1908)

*Synonym.* *S. canthel* Castellani, 1908

children in the tropics

FAMILY 2 OÖSPORACEÆ Saccardo, 1886

**Definition.**—Blastosporineæ in which the hyphæ may be long or little different from the spores, which are typically in chains

**Classification.**—This family is divided into several genera—e.g. *Oospora* Wallroth, 1833 *Monilia* Persoon 1797 and *Oidium* Link 1809 which may be distinguished as follows—

A Hyphæ thin short simple or nearly simple, terminating in chains of spores—*Oospora*

B Hyphæ not thin often long and branched—

I Sporophores simple or subsimple typically with disjunction apparatus Glucose completely fermented gas being produced Numerous budding forms in cultures—*Monilia*

II Sporophores simple septate often with disjunction apparatus Glucose not completely fermented gas not being produced Budding forms rare in cultures—*Oidium*

These genera may now be briefly described

Genus *Oospora* Wallroth 1833

**Definition.**—Oösporaceæ with a lax or compact mycelium in which nuclei are differentiated nearly simple. The conidia are or brightly coloured are arranged regularly in chains

**Remarks.**—Saccardo in his 'Sylloge Fungorum' recognizes a large number of species as belonging to this genus, and these are grouped into sections by the varying colour of the conidia

The only species known to cause disease in animals referable to this genus is *Oospora canina* Sabrazes 1893 which causes favus in dogs and which can produce an eruption resembling ringworm when inoculated into man. According to Sabouraud however it has never been known to cause disease in man spontaneously (i.e., without experimental inoculation), and is therefore of little importance at present

Vuillemin considers that *Achorion schoenleinii* Lebert, 1845, the fungus of favus of human origin, belongs to this genus

was wanting that it was the causal organism although its association was intimate

### Genus *Pityrosporium* Sabouraud, 1895

Synonym.—*Dermatophyton* Dodd, 1910

Definition.—Cryptococcaceae without well developed double contour

Type Species—*Pityrosporium ovale* (Bizzozzero 1882)

Remarks and Classification.—This genus which is difficult to classify is allied in appearance in some forms to a budding yeast, and as such comes close to *Cryptococcus*. No species has so far been cultivated (Dodd claims to have cultivated *P. malassези*).

children in Ceylon

They may be distinguished as follows —

- A Flask-shaped or oval 3 to 15 microns but usually small—*Ovale*
- B Generally roundish 5 to 16 microns but usually large—*Cantliei*



FIG 560 — *Pityrosporium ovale*  
BIZZOZZERO



FIG 561 — *Pityrosporium ovale*  
BIZZOZZERO

(After Sabouraud)

### *Pityrosporium ovale* (Bizzozzero, 1882)



is said to have lateral as well as terminal conidia and is classified here as a cryptococcus

The genus *Parasaccharomyces* de Beurmann and Gougerot 1909 with its species *Parasaccharomyces larteri* Verdun 1912 found in a case of enteritis with hepatic bronchial and cutaneous lesions has not merely yeast like forms but septate hyphæ but it is not known whether it develops terminal conidia in chains and therefore it may at present correctly be placed in the genus *Cryptococcus* until more is known about it and the same remarks apply we think to *Parendomyces* Querat and Laroche 1909



Fig 564

Fig 565

Fig 566

FIG 564 — *Monilia zeylanica* CASTELLANI GLUCOSE AGAR CULTURE

FIG 565 — *Monilia brodiaei* CASTELLANI GLUCOSE AGAR CULTURE

FIG 566 — *Monilia nuxea* CASTELLANI GLUCOSE AGAR CULTURE

**Remarks**—A number of species belonging to this genus are known to exist in nature growing on decomposing wood dead leaves and fruits

The number of species known to infect man has been considerably extended during recent years by Castellani. The *Monilias* are of importance in that they are considered to be the aetiological agents of thrush bronchomycosis (*pro parte*) some dermatomycoses and according to certain authors of sprue

**Type Species**—It is difficult to know which is really the type species of this genus but probably it is *Monilia aurea* (Link 1791)

Genus *Monilia* Persoon 1797

Definition.—Vogne

Original Definition.—*Stipitata aut effusa byssoidea Fila moniliformis articulata*

thallospores of the blastospore type are formed. Glucose and often other carbohydrate media fermented with the production of gas.



FIG. 562.—*Monilia tropicalis* CASTELLANI. FRESH PREPARATION FROM SPOTUM.

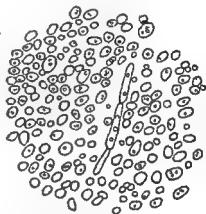


FIG. 563.—*Monilia intestinalis* CASTELLANI. PREPARATION FROM GLUCOSE AGAR CULTURE.

**Nomenclature**—A few words are necessary with regard to the nomenclature and the synonyms as the greatest confusion has existed with regard to the genus.

teenth edition) of Linnæus's "Systema Naturæ" 1791 refers it is true to *Monilia aurea* (Link 1791) but it was described as *Oidium aureum* Link 1791. The other synonyms do not require explanation.

We have not included the genus *Zymonema* de Beurmann and Gougerot 1909 in the synonyms because *Z. gilchristi*, the cause of American blastomycosis more usually called *Cryptococcus gilchristi*,

TABLE SHOWING PRINCIPAL BIOCHEMICAL AND CULTURE REACTIONS OF THE FUNGI IN ALPHABETICAL ORDER

Genus Mon	L. truss M k	Glu co e	Levu lose	Val toe.	G lac to n	Saccha ro e.	Lac tose	Man nise	Dul c e.	Pe tr
I alba Castellani	AC	AG	AG	AG	AG	A	O	O	O	O
I albicans Robin, emd Cast	AC	AG	AGs	AGs	AG	Avs	O	O	O	O
I balcanica Cast	O	AG	As	O	O	O	O	O	O	O
I blanchardi Cast	Avs V k	AGs	A	A	A	A	O	O	O	O
I bronchialis Cast	O	AG	AG	AG	O	AGs	O	O	O	O
I burgessi Cast	O V k	AGs	A	AGs	A	AGs	O	O	O	O
I chalmersi Cast	As V k	AG	AG	As	AGs	AG	O	O	O	O
I decolorans Cast and Low	DC	AG	AG	AG	A	A	O	O	O	O
I enterica Cast	O V k	AG	AG	AG	AG	AG	O	As	O	O
I faecalis Cast	A DPs	AG	AG	AG	AGs	AGs	O	O	O	O
I guillermondi Cast	O V k	AG	AG	As	A	AG	O	O	O	O
I insolita Cast	As V k	AG	AG	AG	AG	AG	O	As	O	O
I intestinalis Cast	ADs	AG	AG	As	A	A	O	O	O	O
I krusei Cast	O	AG	AG	O	O	O	O	O	O	O
I londinensis Cast	AC	AG	AG	A	A	A	A	O	O	O
I lustigi Cast	As D	A	AGs	Avs	A	AGs	O	O	O	O
I macedoniensis Cast	AC	AG	AG	AorO	AG	AG	O	O	O	O
I metalondinensis Cast	O	AG	AG	AG	AG	O	O	O	O	O
I metatropicalis Cast	AC	AG	AG	AG	AG	AG	O	O	O	O
I nabarroii Cast	AC	AG	AG	AG	O	O	O	O	O	O
I negrii Cast	As alk	AG	AG	As	AGs	AG	O	O	O	O
I nitida Cast	A DC	AG	AG	A	A	A	A	A	O	O
I nivea Cast	O V k	AG	AG	AG	AG	AGs	O	O	O	O
I parabalcanica Cast	AC	AG	As	O	O	O	O	O	O	O
I parichalmersi Cast	AC	AG	AG	O	AG	AG	O	O	O	O
I parakrusei Cast	AC	AG	AG	O	O	O	O	O	O	O
I paritropicalis Cast	As alk	AG	AG	AG	AG	AG	O	O	O	O
I perryi Cast	As D V k	A	AGs	A	A	AGs	O	O	O	O
I vinoyi Cast	O	AG	AG	AG	O	O	O	O	O	O
do bronchialis Cast	AC	AG	AG	AG	O	AG	O	O	O	O
do guillermondi Cast	AC or P	AG	AG	O	O	AG	O	O	O	O
do londinensis Cast	O	AG	AG	AG	AG	O	O	O	O	O
I pseudo londinoides Cast	AC	AG	AG	AG	AG	O	O	O	O	O
I pseudo metalondinensis	AC	AG	AG	AG	AG	O	O	O	O	O
I pseudo-tropicalis Cast	ACs	AG	AG	O	AGs	AG	AG	O	O	O
I pulmonalis Cast	O alk D	AG	AG	AG	AGs	AG	O	Avs	O	O
I rhoi Cast	As A k	AG	AG	Avs	AGs	AG	O	O	O	O
I rosea Zenoni	—	—	—	—	—	—	—	—	—	—
I tropicalis Cast	A	AG	AG	AG	AGs	AGs	O	O	O	O
I zeylanica Cast	ACs	A	A	A	A	A	As	O	O	O

have no serum produce a dium all round the growth using the serum of *Bos indicus*. The biochemical properties and sugar reactions vary from species to species. Rabbits can be immunized for these fungi and attempts to use the immunization and agglutination reactions in the differentiation of the various species have been made with only partial success as there is a large production of co agglutinins in addition to the specific ones for very different fungi—*e.g.* fungi of the genus

CONCLUSION IN THE CLASSIFICATION OF SUCH FUNGI. Many of the species of this genus can hardly be distinguished by purely morphological and botanical characters. We are of opinion that the classification should be based not only on morphological data but also on biochemical characters and immunization phenomena. Among the

the reactions with certain sugars are constant while with others (for instance mannite) these may vary and the fermentative power on certain sugar—*e.g.* galactose—may be rapidly lost. Hence species should be compared only in strains recently isolated. It is to be noted also that in analogy to intestinal bacteria a species may be trained to ferment certain sugars on which it did not yet

classified into its group it is easy by means of the table on pp. 1082-1083 to make the specific determination—

- |   |   |   |
|---|---|---|
| A | Gas produced in glucose only—Group I          | <i>Filantica</i> group— <i>M. balcanica</i> , <i>M. parabalcanica</i> |
| B | Gas produced in glucose and levulose—Group II | <i>Armitis</i> group—   |
| C | " " " " " " " " " " " "                       | Group III <i>Pinosy</i>   |
| D | " " " " " " " " " " " "                       | Group IV <i>pseudomata on d nensis</i>                                |





lium These globular elements (conidia) become detached and reproduce by budding

**Cultures**—The fungus grows best of all on slightly acid Sabouraud's maltose agar or on glucose agar but grows fairly well also on all alkaline media In cultures the fungus appears under two forms—a globular form and reproducing by threads simple or

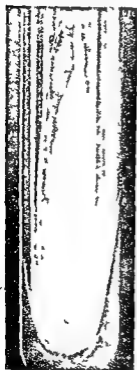


FIG 568—*Monilia tropicalis* CASTELLANI

asci absent. On Sabouraud's and glucose agar the growth is abundant smooth of a creamy white colour on ordinary agar the growth is less abundant Gelatine and serum are very slowly liquefied Milk is rendered acid and after a time it clots The sugar reactions are given in the table

**Pathogenicity.**—*M. albicans* is one of the fungi which gives rise to thrush This condition may be due to several different fungi—*M. albicans* *Endomyces oullemism*

#### *Monilia tropicalis* (Castellani 1909)

**Synonym.**—*Endomyces tropicalis* Castellani 1909

Found by Castellani in Ceylon in many cases of bronchomycosis In the expectoration round or oval shape yeast like cells are seen and at times segments of mycelium Cultures are easily obtained on Sabouraud's or glucose agar and even on ordinary agar It grows more abundantly on slightly acid than on alkaline media On Sabouraud's and glucose agar the growth is abundant creamy white with a smooth surface when young, often

slightly crinkled when old The growth is composed practically of only globular yeast like cells while in the water of condensation globular cells and mycelium may be found together A little mycelium may be found however, occasionally in the growth on the slope Ascus formations are absent Gelatine and serum are not liquefied

as is Litm

sugar reactions will be found in the table on pp 1082 1083 It produces acid and gas in glucose, levulose, maltose, and also, in less

C *Species found in vaginal discharge in the tropics and in Europe* —

D *Species found in man, but not classified* —

1 *M. lactea* Castellani 1913

2 *M. luteicolor* Castellani 1913

A brief description of these species may be given

***Monilia albicans* Robin, 1853**

1853, *Syringospora robiniana* Rees, 1877, *Monilia* Vuillemin, 1898

past used to cover a number of



FIG 567 — *Monilia albicans* ROBIN

out. As the species *M. albicans* has to be split into many species we keep the term *M. albicans* for the species which we are now

and  
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bent  
ngth



lum These globular elements (conidia) become detached and reproduce by budding

**Cultures**—The fungus grows best of all on slightly acid Sabouraud + maltose agar or on glucose agar but grows fairly well also on all alkaline media In cultures the fungus appears under two forms—a globular form morphologically similar to a typical yeast and reproducing by budding, a filamentous form showing mycelial threads simple or ramified *Asci* and *internal spores* as in the genus *Endomyces*, are absent. On Sabouraud's and glucose agar the growth is abundant, smooth of a creamy white colour, on ordinary agar the growth is less abundant Gelatine and serum are very slowly liquefied Milk is rendered acid and after a time it clots The sugar reactions are given in the table

**Pathogenicity.**—*M. albicans* is one of the fungi which gives rise to thrush This condition may be due to several different fungi—*M. albicans* *Endomyces vullemini* *M. tropicalis* etc (p 1741) Thrush is generally restricted to the oral mucosa but in certain cases it may spread to the oesophagus stomach intestine *M. albicans* has been found also in a few cases of bronchomycosis

#### *Monilia tropicalis* (Castellani, 1909)

**Synonym.**—*Endomyces tropicalis* Castellani 1909

Found by Castellani in Ceylon in many cases of bronchomycosis In the expectoration round or oval shape yeast like cells are seen and at times segments of mycelium Cultures are easily obtained on Sabouraud's or glucose agar and even on ordinary agar It grows more abundantly on slightly acid than on alkaline media On Sabouraud's and glucose agar the growth is abundant creamy white with a smooth surface when young, often

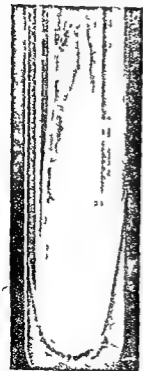


FIG 568—*Monilia tropicalis* CASTELLANI

slightly crinkled when old The growth is composed practically of only globular yeast like cells while in the water of condensation globular cells and mycelium may be found together A little mycelium may be found however occasionally in the growth on the slope *Asci* formations are absent Gelatine and serum are not liquefied, there is no brownish discoloration of the serum

by Castellani also in a few cases of *thrush* and in a case of *pseud*

**Monilia paratropicalis** (Castellani 1909)

*Monilia paratropicalis* Does not coagulate milk. Differs from the type *M. tropicalis* by producing acid and gas in very large amount in succharose and in rendering dextrin acid (see table)

The same fungus or a very similar one was found in two cases of blastomycosis of the skin in Ceylon

**Monilia pseudo-tropicalis** (Castellani 1910)

Reactions see table

**Monilia metatropicalis** Castellani 1916

Differs from *M. tropicalis* in clotting milk

**Monilia bronchialis** (Castellani 1910)

66

1 on milk

**Monilia chalmersi** Castellani 1912

in the table

**Monilia para-chalmersi** (Castellani 1917)

Differs from *M. chalmersi* in slowly liquefying gelatine and in other minor characters. Found in sputum

**Monilia macedoniensis** Castellani 1917

Found in sputum. Clots milk. Belongs to the Chalmersi group

**Monilia guillermondi** (Castellani 1910)

**Synonym** — *Endomyces guillermondi* Castellani 1910  
 Observed in alkaline Sert actions see table  
 Milk is rendered For sugar re-

**Monilia pseudo-guillermondi** Castellani 1916

Clots milk

**Monilia nivea** (Castellani 1910)

**Synonym** — *Endomyces niveus* Castellani  
 Found in a sample of sputum which had not been collected in a sterile vessel Of doubtful pathogenicity For cultural and biochemical characters see table

**Monilia nitida** (Castellani 1910)

**Synonym** — *Endomyces nitidus* Castellani 1910  
 Observed in a sample of sputum by Castellani Of doubtful pathogenicity Milk is rendered first acid then decolourized and clotted Other cultural characters are found in the table

**Monilia zeylanica** (Castellani 1910)

**Synonym** — *Endomyces zeylanicus* Castellani 1910  
 Found in sputum by Castellani Growth on glucose agar of a yellowish colour Milk is rendered very acid and is slowly clotted Gelatine and serum are not liquefied Does not produce gas in any carbohydrate with the doubtful exception of raffinose

**Monilia krusei** (Castellani 1909)

**Synonyms** — *Saccharomyces krusei* Castellani ; *Endomyces krusei* Castellani

Found in sputum by Castellani In sputum it appeared as a *saccharomyces* and in cultures had all the characters of a *saccharomyces* at first except that no asci were observed after several days as present on Sabouraud's agar the table It produces acid and reactions remain constant after

ten years

**Monilia pinoyi** (Castellani 1910)

**Synonym** — *Endomyces pinoyi* Castellani 1910  
 Found in sputum Grows well on Sabouraud's and glucose agar and other sugar media also on ordinary agar though less abundantly Does not clot milk does not liquefy either gelatine or serum Produces acid and gas in glucose levulose maltose

**Monilia enterica** (Castellani 1911)

**Synonym** — *Endomyces entericus* Castellani *Monilia psilostis* Castellani

**Monilia paratropicalis** (Castellani 1909)

**Synonym**—*Endomyces paratropicalis* Castellani 1910

Found in some cases of bronchomycosis by Castellani Microscopically and on Sabouraud's and glucose agars identical with *M. tropicalis*. Does not coagulate milk. Differs from the typical *M. tropicalis* by producing a smaller amount of gas.

two cases

**Monilia pseudo-tropicalis** (Castellani 1910)

Differs from *M. tropicalis* in the production of gas. For the sugar reactions see table.

**Monilia metatropicalis** Castellani 1916

Differs from *M. tropicalis* in clotting milk.

**Monilia bronchialis** (Castellani 1910)

**Synonym**—*Endomyces bronchialis* Castellani 1910

Found in sputum. Colonies white. Has no action on milk or gelatine serum. Sugar reactions are found in the table.

**Monilia chalmersi** Castellani 1912

in the table

**Monilia parachalmersi** (Castellani 1917)

Differs from *M. chalmersi* in slowly liquefying gelatine and in other minor characters. Found in sputum.

**Monilia macedoniensis** Castellani 1917

Found in sputum. Clots milk. Belongs to the Chalmersi group.

mass (generally embedded in wax) in which numerous long septate mycelial threads  $\frac{1}{2}$  to  $3\frac{1}{2}$   $\mu$  in breadth and numerous roundish free spores  $2\frac{1}{2}$  to  $3$

it {  
doi  
in

In the cases of otomycosis the mycelium was abundant the mycelial threads being very long and septate  $3$  to  $4$   $\mu$  in breadth very numerous free round spores  $4$  to  $5$   $\mu$  in diameter were present For cultural characters see table

#### *Monilia burgessi* (Castellani 1912)

Synonym — *Endomyces burgessi* Castellani 1911

Isolated from the air Grows abundantly on the usual sugar media the growth being of a white creamy appearance Does not render acid or coagulate milk Does not liquefy serum or gelatine On serum it produces a zone of peculiar brownish or black discoloration in the medium all round the growth The sugar reactions are found in the table

#### *Monilia pulmonalis* Castellani 1911

Found by Castellani in sputum and also in samples of tea For cultural characters see table (p 187)

#### *Monilia lustigi* Castellani 1912

Found in samples of tea Grows well on all sugar media and also on ordinary agar the growth being of a snow white colour Renders litmus milk slightly acid and then decolorizes it completely Does not liquefy serum or gelatine On serum it induces a narrow zone of black discoloration all round the growth The sugar reactions are found in the table (p 1082)

#### *Monilia balcanica* Castellani 1916

Found in sputum and also in a case of dermatitis of which it was not the cause Produces gas in glucose only Levulose often rendered acid (see table)

#### *Monilia parabalcanica* Castellani 1916

Differs from *M. balcanica* in clotting milk

#### *Monilia perryi* Castellani 1912

Found by Castellani in samples of tea dust The cultural characters and chemical properties are seen in the table

#### *Monilia narbarroi* Castellani 1917

Clots milk and produces gas in glucose levulose and maltose Found in sputum, An identical or very similar variety has been found in vaginal mucus by Castellani and Taylor

*Monilia faecalis* (Castellani 1911)

Synonym — *Endomyces faecalis* Castellani 1911

Found in intestinal contents by Castellani Milk is rendered  
slightly acid then alkaline Gela  
medium is not liquefied ugar re

*Monilia insolita* (Castellani 1911)

Synonym — *Endomyces insolitus* Castellani 1911

Found in stools and in the saliva The milk is rendered first  
slightly acid then alkaline  
develops on the surface of  
medium is not liquefied I  
table

*Monilia intestinalis* (Castellani 1911)

Found in intestinal contents by Castellani 1911

*Monilia alba* (Castellani 1911)

Clots milk Produces acid and gas in glucose levulose maltose  
galactose and acid only in saccharose Does not liquefy gelatine  
or serum

*Monilia rhei* (Castellani 1909)

Synonym — *Endomyces rhei* Castellani 1909

Found by Castellani in Ceylon in several cases of otomycosis  
and once in a sample of tea In the ear the fungus forms a whitish

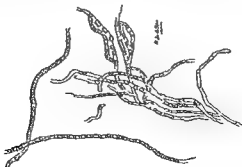


FIG. 569.—*Monilia rhei* CASTELLANI

(From a fresh preparation. The free spores are not shown.)



**Monilia blanchardi** (Castellani, 1912)

**Synonym**—*Endonyses blanchardi* Castellani 1912

Isolated from tea dust by Castellani. Grows abundantly on maltose glucose and other sugar media also on ordinary agar though less vigorously. The growth is of a white colour and smooth surface. Milk rendered at first very slightly acid and then alkaline. No liquefaction of gelatine or serum. Does not produce gas in any sugar except in small quantity in glucose.

**Monilia bethaliensis** Piper 1918

Found by Piper in a case of bronchitis. Ferments glucose and maltose with production of gas. Action on levulose not known. Gelatine not liquefied, milk not clotted.

**Monilia rosea** (Zenoni, 1912)

**Synonym**—*Cidium roseum* on liquefaction Zenoni, 1912

pigs and white rats

**Monilia subtilis** (Blanchard, 1895)

**Synonyms**—*Cidium subtilis cutis* Babès 1895 *Mycoderma subtilis* Veidun 1912

Found by Babès in some ulcers on which the fungus produced white membrane. The membrane was white and soft.

**Monilia pulmonea** (Bennett, 1842)

**Synonyms**—*Cidium pulmonea* Bennett 1842 *Oospora pulmonea* Sa cardo 1886 *Mycoderma pulmonea* Vuillemin 1891

The organism was found in a case of pneumothorax by Bennett in 1842, it was observed again by Vuillemin in the sputum of a tubercular patient in 1891 and in an ulcerative dermatitis by Balzer, Burnier and Gougerot in 1910. It grows under two types—a saccharomyces-like and a filamentous type. Colonies on glucose agar white. Biochemical reactions not given.

**Monilia candida** (Bonorden, 1851)

**Synonym**—*M. bonordeni* Vuillemin

Commonly found in ulcers, but not in any other form. It is occasionally



Remarks—This fungus was isolated from stools in cases of sprue and from the sputum in cases of chronic bronchitis

*Oldium matalense* Castellani 1915

Definition—*Oldium* producing a white growth on glucose agar. It turns milk sometimes slightly acid without the formation of a clot or it may have no effect. Gelatine is not liquefied nor is any gas produced in carbohydrate media.

FAMILY 3 ENANTIOTHAMNACEÆ Chalmers and Archibald 1915

Thallus  
lar septa  
the seg

ments which are 2-2.5 microns broad. The conidia are arranged verticillately around the septa.

Remarks—This family contains only one genus *Enantiothamnus* Pinoy 1911. The name is derived from *εναντιος* opposite and *θαμνος* a shrub.

Genus *Enantiothamnus* Pinoy 1911

Definition—*Enantiothamnaceæ* with the characters of the family.

Remarks.—There is only one species so far known *Enantiothamnus brauli* Pinoy 1911.

*Enantiothamnus brauli* Pinoy  
1911

Definition—*Enantiothamnus* with oval conidia 2.25 by 1.15 microns.

Habitat—Parasitic in man.

Remarks—This fungus was discovered by Brault in umbilicated tumours containing pus in the gluteal region of an Arab in Algiers.

On Sabouraud's agar the colonies are white with a yellowish central portion.

Pathogenicity—Produces tumours and pus in man and is pathogenic for guinea pigs.



FIG 577—*Enantiothamnus brauli* Pinoy  
(After Brault and Pinoy)

FAMILY 4 HAPLOGRAPHIACEÆ Saccardo 1896

Definition—*Blastosporineæ* with hyphæ manifest and distinct from the conidia which are usually arranged in chains or in parasitic condition in man in grape-like masses.



FIG 576—*Oldium matalense* CAS  
TELLANI  
CUL  
TURE

Remarks—It was found by Link in milk and recently by Linossier in cases of bronchitis. The latter was considered to be slightly different and to form a pathogenic race *Oidium lactis* var A.

*Oidium rotundatum* Castellani 1911

Definition—*Oidium* growing on glucose agar and producing crinkled or vermiform yellowish growths. It gives rise to acidity in milk and after a variable time may form a clot. Gelatine is



Fig 573

Fig 574

FIGS 573 AND 574—*Oidium rotundatum* CASTELLANI CULTURE



FIG 575—*Oidium asteroides* CASTELLANI CULTURE

liquefied but liquefaction may be extremely slow or absent with certain strains. For the sugar reactions see the table (p 1094). It

ar or

*Oidium asteroides* Castellani 1914

Definition—*Oidium* producing colonies with a characteristic radiating appearance on glucose agar from which it derives its name. It grows badly or not at all upon inspissated blood serum which it does not liquefy. It turns milk acid and clots it in a variable time. No gas is formed in any carbohydrate medium.



FIG 578—*Malassezia tropica* CASTELLANI  
(From a fresh preparation in liquor potassa Old case)



FIG

(From a fresh

are in



Genus *Cladosporium* Link 1816

**Definition.**—Cladosporiaceæ with decumbent hyphæ Conidiophores bearing smooth uniform conidia arranged in short chains or solitary

**Etymology.**—The name is derived from κλαδος 'a young shoot'

**Remarks**—The type species is *Cladosporium herbarium* Persoon 1801 and there are a very large number of species scattered all over the world and commonly found on plants in tropical gardens from whence the spores can easily be conveyed to the human skin and either grow the scales pure culture causal organisms but they do not agglutinate with the patient's serum

They may also grow as contaminations of laboratory media so commonly may their spores be found in the air The two species known in man may be recognized as follows —

Habitat tinea nigra—Species *Cladosporium mansonii* (Castellani 1908)

Habitat ulcerating nodules—Species *Cladosporium penicilloides* Gueguen 1911

*Cladosporium mansonii* Castellani 1905

**Synonyms**—*Microsporium mansonii* Castellani 1905 *Foxia mansonii* Castellani 1908 *Cladosporium mansonii* Pinoy 1912

the are  $\frac{1}{2}$  to  $3\frac{1}{2}$   $\mu$  in diameter non tinted Sometimes they may be irregular in outline bent banana shaped The spores are globular and most of them very large—5 to 10  $\mu$  They are frequently arranged in clusters

The fungus is easily cultivated by inoculating scrapings of the affected patches on maltose agar

colonies may remain separate or more often gradually coalesce into a jet black knobby mass deeply rooted into the medium



FIG 582—*Cladosporium mansonii* CAS  
TELLANI YOUNG  
AGAR CULTURE

*Malassezia furfur* Ch Robin 1853

**Synonyms**—*Microsporon furfur* Ch Robin 1853 *Sporotrichum furfur* Saccardo 1886 *Malassezia furfur* Baillon 1889 *Oidium furfur* Zopf 1890 *O. subtile* Kothar 1892

Mycelium abundant septate non ramified some mycelial threads are much larger than others the breadth varies between 3 and 4  $\mu$ . The spores are roundish (3 to 5  $\mu$  in diameter) and run into clusters. Attempts at cultivation have failed. It is the cause of pityriasis versicolor.

*Malassezia tropica* Castellani 1905

with numerous  
shape spores  
with a double  
collected in

clusters. The fungus does not grow on artificial media. It is the cause of tinea flava or pityriasis flava of tropical climates.



FIG 580—*Cladosporium mansoni* CASTELLANI  
(From a preparation stained by fuchsin)



FIG 581—*Cladosporium mansoni* CASTELLANI HANGING-DROP CULTURE

## FAMILY 5 CLADOSPORIACEÆ Saccardo 1886

Conidia smooth not capitate more or less in chains at first. Hyphae and conidia uniform. Hyphae not inflated but decumbent conidia in short chains and finally solitary.—Census *Cladosporium* Link 1809

Genus *Cladosporium* Link 1816

**Definition**—Cladosporiaceæ with decumbent hyphæ Conidophores bearing smooth uniform conidia arranged in short chains or solitary

**Etymology**—The name is derived from κλαδος a young shoot

**Remarks**—The type species is *Cladosporium herbarium* Persoon 1801 and there are a very large number of species scattered all over the world and commonly found on plants in tropical gardens from whence the spores can easily be conveyed to the human skin and either grow there causing a lesion or simply remain sheltered among the scales of other lesions from which they may be grown in pure culture thus giving rise to the impression that they may be causal organisms but they do not agglutinate with the patient's serum

They may also grow as contaminations of laboratory media so commonly may their spores be found in the air The two species known in man may be recognized as follows —

Habitat tinea nigra—Species *Cladosporium mansonii* (Castellani 1908)

Habitat ulcerating nodules—Species *Cladosporium penicilloides* Gueguen 1911

*Cladosporium mansonii* Castellani 1905

**Synonyms**—*Microsporium mansonii* Castellani 1905 *Foxia mansonii* Castellani 1908 *Cladosporium mansonii* Pinoy 1912

The fungus is found very abundantly in the lesions of tinea nigra the mycelial articles are rather short—18 to 20  $\mu$  in length and 2½ to 3½  $\mu$  in breadth non ramified Sometimes they may be irregular in outline bent banana shaped The spores are globular and most of them very large—5 to 10  $\mu$  They are frequently arranged in clusters

The fungus is easily cultivated by inoculating scrapings of the affected patches on maltose agar After two to six days



**Cladosporium penicilloides** Gueguen 1911

**Synonym** — *C. Madagascariense* Verdun 1913

Found by Fontoynt in a patient with some ulcerated nodules of the leg in Madagascar. The fungus grows easily on Sabouraud and other media giving rise to black cerebriform colonies. In hanging drop cultures shows the typical features of the genus.

*Cladosporium herbarium* Iersoon has been demonstrated by Nasse to be the cause of the black spots so often found on imported frozen meat.

## SUBORDER 2 ARTHROSPORINEÆ VUILLEMIN 1910

**Definition** — Thallospores with yeast like forms associated in

A Producing *Piedra* on hairs — Genus *Trichosporum* Behrend 1890

B Producing *Black Maduromycosis* — Genus *Madurella* Brumpt 1905

C Producing *White Maduromycosis* — Genus *Indiella* Brumpt 1906

**Genus Trichosporum** Behrend 1890.**Definition**

in the form of  
substance

**Remarks**

Behrend 1890 with *Trichosporum* Fries 1849 a very different genus with over forty species mostly saprophytic

The species of this genus give rise to nodosities on hairs. They are —

*T. giganteum* Behrend 1890 the cause of *piedra* in Columbia in hairs of the head

*T. beigelii* (Rabenhorst 1867) the cause of *piedra* in Europe in hairs of the beard

*T. ovoides* (Behrend 1890) the cause of *piedra* in hairs of the moustache



I  
T  
T

- A *Bodies around hair polyhedral* —  
 I Diameter of bodies 12-15 microns—*Giganteum*  
 II Diameter of bodies 3-4 microns—*Beigelii*
- B *Bodies around hair oval and small* 3-4 microns by 1.5-2.5 microns —  
 I In cultures hyphae often twisted like a corkscrew—*Ovale*  
 II In cultures hyphae not so twisted—*Ovoides*
- C *Bodies around hair roundish* 3-4 microns in diameter —  
 Fungus associated with a coccus with which it grows well on sugar media—*Glycophile*

These fungi live parasitic on the surface of the hairs but do not penetrate into their interior during their parasitic life they vegetate

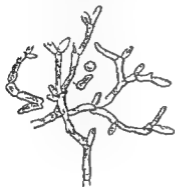


FIG 583—TRICHOSPORUM  
(After Vuillemin)

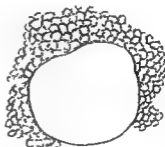


FIG 584—TRANSVERSE SECTION  
THROUGH A PIEDRA NODULE

tate in the shape of large oval or roundish elements embedded in an amorphous substance. Saprophytically (cultures) they vegetate forming mycelial threads and spores.

### *Trichosporum giganteum* Behrend 1890

This is the cause of *piedra* of Columbia develops on the surface of the hair in the shape of large polyhedral cells 12 to 15  $\mu$  in diameter. Masses of the fungus form hard nodules along the hair. The fungus is easily grown on various media. In cultures the mycelial threads are septated cylindrical between 1 and 4  $\mu$  wide. The spores are of various dimensions between 3 and 12  $\mu$ . The colour of the colonies is light brownish. Horta describes in the nodules of a variety of *piedra* certain large cyst-like bodies containing generally eight fusiform bodies. When the membrane bursts these bodies escape being provided with one flagellum at each end. Pinoy is inclined to consider these formations to be asci containing cubated ascospores.

The optimum temperature for the growth of the fungus is between 30° C and 32° C above 35° C and under 25° C the growth is much slower and may be nil under 20° C

This fungus is the cause of tinea nigra (p. 20, 8)

### Cladosporium penicilloides Gueguen 1911

See also *Cladosporium penicilloides* Gueguen 1911

*Cladosporium herbarium* Persoon has been demonstrated by Nassee to be the cause of the black spots so often found on imported frozen meat

## SUBORDER 2 ARTHROSPORINEÆ VUILLEMIN 1910

**Definition**—Thallosporales with yeast like forms associated in cultures with hyphæ and other forms with longer hyphæ. Reproduction by means of arthrospores parasitic on man

Trichosporium, Madurella, Indrella

recently been supported by the work of Marshall and one of us on *T. curvis*. However for the present we propose to leave these genera and their allies out of consideration

**Classification**—This suborder contains the following genera—

A Producing *Piedra* on hairs—Genus *Trichosporium* Behrend 1890

B Producing *Black Maduromycosis*—Genus *Madurella* Brumpt 1905

C Producing *White Maduromycosis*—Genus *Indrella* Brumpt 1906

### Genus *Trichosporium* Behrend 1890

**Definition**  
in the form of  
substance

**Remarks**—  
Behrend 1890 with *Trichosporium* Fries 1849 a very different genus with over forty species mostly saprophytic

The species of this genus give rise to nodosities on hairs. They are—

*T. giganteum* Behrend 1890 the cause of *piedra* in Columbia in hairs of the head

*T. bergeli* (Rabenhorst 1867) the cause of *piedra* in Europe in hairs of the beard

*T. ovoides* (Behrend 1890) the cause of *piedra* in hairs of the moustache

Genus *Madurella* Brumpt 1905 *emendavit* Pinoy 1912

**Definition** — Arthrosporineæ with sterile septate hyphæ reproducing the thallus by fragmentation and secreting a black pigment. The spores are produced secondarily by binary division of the articles. Found in black maduromycosis and grow well at 37° C.

**Type Species** *M. mycetomi*

**Historical** —  
account of a  
Djibouti. In

1819 wrote an  
observed at  
in this case

sent from Madagascar



FIG 586 — *Madurella mycetomi*  
LAVERAN

(After Brumpt)



FIG 587 — GRAINS OF *Indella reyniersi* BRUMPT

Bouffard in 1905 reported the presence of the same disease in Senegal and in the French Sudan.

In this variety the grains are black or deep brownish red and always hard and generally small from 1-2 millimetres in diameter when single and not in accumulated masses. The surface is irregular with projecting points. On clearing with Eau de Javelle

*Madurella* for this fungus

(bo  
mer  
m c  
secr  
the  
scler  
diameter (chlamyospores)

The type species is the organism called *Streptothrix mycetomi* by Laveran in 1902 which therefore becomes *Madurella mycetomi* (Laveran 1902) first cultivated by Brault (1911) in material from Algerian cases.

This form of mycetoma was reported by Balfour (1911) to be present in the Anglo Egyptian Sudan.



Old cultures on glucose agar or on glycerinated glucose agar were quite different in the two species

On carrot *M. tozeuri* attained a deeper brownish yellow colour while in old cultures on this medium it produced spores in a manner resembling an *Oospora*

Pinoy in his remarks upon the mycology of these two species says that Brault's *M. mycetomi* very closely resembles that isolated

they arise varying from 2.5 microns while the membrane becomes

4 x millimetre in diameter and are composed of hyphal segments more or less cylindrical. Sometimes the sphere attains a diameter of 10 microns and usually contains only one nucleus but though studied for a long time these sclerotes were never observed to have any higher form of fructification. In *M. tozeuri* it is very rare to see the formation of sclerotes which takes place on the surface of the medium.

On the bases of the researches on *M. mycetomi* and *M. tozeuri* Pinoy classifies the genus *Madurella* as follows —

- A Sclerotes 0.5 x millimetre in diameter formed in the depths of medium in cultures—*Mycetomi*
- B Sclerotes rarely produced and then on the surface of the medium—*Tozeuri*

#### *Madurella mycetomi* (Laveran 1902)

Synonym — *Streptothrix mycetomi* Laveran 1902

Mycelium greyish white when old yellowish and darkening the media in sugar cultures. Spores varying in dimension from 2.5 microns. Sclerotes black and sterile with a diameter from 0.5 x millimetre formed in the depths of the medium in cultures. Can invade the skin, bone, muscles and connective tissue of man giving rise to black grains which are small, hard, round and more or less warty and which morphologically resemble the sclerotes formed in the cultures. Up to the present the inoculation into animals is negative. Very widely spread in Africa. Isolated by Brault from a mycetoma with black grains in Algeria.

#### *Madurella tozeuri* (Nicolle and Pinoy 1908)

Synonym — *Obspora tozeuri* Nicolle and Pinoy 1908

Mycelium white becoming yellowish with age and darkening the medium in sugar cultures. Spores generally small 2 microns or

It is generally assumed that this and the Asian together with the American type are one and the same disease but this still requires proof

In 1908 Nicolle and Pinoy described a maduromycosis which they found in Southern Tunisia near the Oasis of Tozeur with hard

cultures of *A. schoenleinii*. The authors looked upon the organism as belonging to the genus *Oospora* Wallroth 1833 with which Vuillemin considers *Achorion schoenleinii* Lebert 1845 should be classified. Its name therefore became *Oospora tozeurii* (Nicolle and Pinoy 1908).

Inoculation experiments were unsuccessful in the rabbit the guinea pig and the monkey but two successful infections were

1908)

Brault (1911 and 1912) cultivated the fungi *Madurella mycelomatis* and *M. tozeurii*.

The former grew at 20° C. and 37° C. on broth various agars potato carrot and some vegetal liquid media.

In the liquid media the growth appeared as a whitish grey puff ball which later became yellowish or brownish while the medium remained clear and the growth fell to the bottom of the tube.

On solid media it formed a greyish white duvet covered growth which possessed a central button surrounded by a radiation and later when the culture was drier the medium became coloured.

Glycerine agar was best as the growth thereon was luxuriant and when old became yellowish in colour while the medium showed a caramel tinge in its entirety.

Glucose glycerine agar produced a growth of the colour of touch wood. This culture is thrown into black wrinkles producing an appearance seen on some cases.

## ORDER III HEMISPORALES Vuillemin 1910

**Definition**—Hyphae with hyphae thin but more than branched conidiophores ramified in a protoconidium preceded by an annular constriction produced by a brown rigid thickening of the wall. The protoconidium is transformed completely or partially into deuteroconidia but occasionally it elongates forming a new conidiophore or puts out branches which are capable of becoming conidiophores.

**Classification**—There is only one genus *Hemispora* Vuillemin 1906

Genus *Hemispora* Vuillemin 1906

**Definition**—Mycelial filaments thin hyaline septated ramified. Each conidiophore terminates into an ampulliform structure (protoconidium) which later divides into several spore-like segments (deuteroconidia).

*Hemispora stellata* Vuillemin 1906

**Definition**—*Hemispora* composed of white sessile discs covered with conidiophores arranged like brown stars in relief on the surface. Deuteroconidia subspherical measuring 2.6-3.5 microns with a dark coloured granular membrane except at the point of attachment sometimes elongated and barrel shaped. Habitat parasitic on man and fungi.

**Remarks**—This species was first found in 1904 by Vuillemin growing on *Aspergillus repens* (De Bary 1870). In 1909 Gougerot and Caraven first found it parasitic in man and this has since been confirmed by other cases described by Auvray De Beurmann Clair and Gougerot and by Thiry. So far it has not been found in the tropics.

**Pathogenicity**—It is the cause of hemisporosis characterized by bony lesions and cold abscesses simulating tertiary syphilis, tuberculosis or sarcoma.

**Biology**—It grows well on sugar media at the temperature of the room and when separated in pure culture can be tested by ment fixation.



FIG. 588.—*Hemispora stellata* VUILLEMIN (After Vuillemin)

*Hemispora rugosa* Castellani 1910

**Synonym**—*Momilia rugosa* Castellani 1910

**Definition**—*Hemispora* growing on all ordinary media with a crinkled surface without asteroid colonies.

**Remarks**—Isolated from cases of bronchitis and a case of tonsillitis by Castellani and recently from a case of thrush by Pijper.

sometimes even 5 microns in diameter. Sclerotes are only rarely produced and then they appear on the surface of the medium

### Genus *Indiella* Brumpt 1906

**Definition.**—Arthroconidium with serrate, ramified branches with

1-8 or 10  $\mu$  septate ramified never secreting—in contrast to the fungi of genus *Madurella*—any black pigment. Masses of mycelial threads form sclerotia like bodies or grains of various shape containing chlamydozooids.

**Classification.**—The species may be recognized as follows—

- A Sclerotia hard and bean shaped—*Mansoni*
- B Sclerotia soft and in coiled masses—*Reynieri*

#### *Indiella mansoni* Brumpt 1906

Mycelial threads are septate, white, thin 1.5-2  $\mu$  when young, thicker (3-5  $\mu$ ) when old. Grains spherical. The fungus has not been grown.

**Pathogenicity.**—It is the cause of Manson's white mycetoma of which only one case is known.

#### *Indiella reynieri* Brumpt 1906

White thallus. Most mycelial filaments are very thin 1-1.5  $\mu$  septate some—those at the periphery—are of irregular shape much broader (4-5  $\mu$ ) when old. Grains spherical. The fungus has not been grown.

of earth worms

**Pathogenicity.**—This fungus causes a variety of mycetoma with white grains (Reynier's white mycetoma) of which there is only one case on record—a European patient who had never left France. The case was observed and reported on by Reynier.



The growth on glucose agar is abundant crinkled or at times somewhat cerebriform and in colour is amber, yellow or brownish. Grows well on gelatine, which it liquefies very slowly so much so



FIG 589—*Hemispora rugosa*  
CASTELLANI 1910 GLU  
COSE AGAR CULTURE



FIG 590—*Hemi-  
spora rugosa*  
GLUCOSE AGAR  
CULTURE



FIG 591—*Hemi-  
spora rugosa*  
GLUCOSE AGAR  
CULTURE

that at first it was believed to be a non liquefier Gram positive certain bottom

#### ORDER IV CONIDIOSPORALES Vuillemin 1910

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ns of

**Remarks**—The reasons for the formation of this order which contains a large number of families have already been given as well as its advantage over the more fully worked out system of Saccardo (p 1037)

**Classification**—The order Conidiosporales is divided into five sub orders which may be recognized as follows —

Name of Fungus	Motility Gram	Gelatine		Serum	Latmus Milk		Lactose	Saccharose	Dulcete	Mannite		Glucose	Maltose
		4	8 12		4	8 12				4	8 12		
Number of Days		4	8 12	4	8 12	4	8 12	4	8 12	4	8 12	4	8 12
<i>Hemispora rugosa</i> Cast	0 +	0 + vs	+	0 0 0	0 0 0	0 0 0	0 0 0	Avs Avs Avs or O or O	0 0 0	0 0 0	0 0 0	A As	As Avs O or Avs

Name of Fungus	Dextrin	Raffinose	Arabinose	Adonite	Inulin	Starch	Salicin	Levulose		Galactose	Glycerin	
								4	8 12			4
Number of Day	4	8 12	4	8 12	4	8 12	4	8 12	4	8 12	4	8 12
<i>Hemispora rugosa</i> Cast	0 0 0	0 0 0	As Avs Avs	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 or Avs Avs	0 0 0	0 0 0	0 0 0

Abbreviations used in the Tables—A=acid, C=cloth, G=gas S=slight, vs=very slight O=negative result—viz non-production of acid in agar media non production of indol non liquefaction of gelatine or serum as the case may be, + =positive result

(a) *Aleuriospore* smooth small acro-pleurogenous —

(b)

### Genus *Acladium* Link 1809

**Definition** — *Aleurismææ* with pale elongate hyphæ and with the mycelium

cernis us—viz

1 *castellani*

### *Acladium castellanii* Pinoy 1916

**Definition** — *Acladium* with small chains of acrogenously placed chlamydospores. The aleuriospores are acropleurogenous.

**Remarks** — The parasite was found by Castellani in cases of ulcerative dermatitis with gumma like nodules in Ceylon the Federated Malay States and Macedonia and fully described by Pinoy.

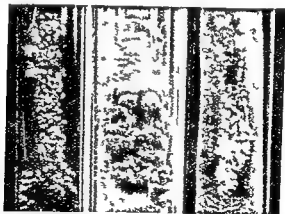


Fig 592

Fig 593

Fig 594

FIGS 592 594 — *Acladium castellanii* PINOY CULTURES ON GLUCOSE AGAR POTATO AND CARROT

The growth on artificial media (such as carrot potato glucose agar) consists of many small roundish masses which later on may coalesce. They are covered by spiculated formations giving them a

A *Conidium imperfect*: Aleuriospore  
Suborder 1: *Aleuriosporineæ* Vuillemin, 1911.

B *Conidium perfect* = *Conidium verum*.

I True Conidiophores absent —

Suborder 2 *Sporotrichineæ* Vuillemin, 1910

II True Conidiophores present —

(a) Conidia borne on sporophores

Suborder 3 *Sporophoralineæ* Vuillemin, 1910

(b) Conidia borne on phialides

1 Prophialides absent

Suborder 4 *Phialidineæ* Vuillemin 1910

2 Prophialides present

Suborder 5 *Prophialidineæ* Vuillemin, 1910

The fourth suborder, Phialidineæ, contains species of the genera



with species parasitic in man

SUBORDER 1 ALEURIOSPORINEÆ VUILLEMIN, 1911

**Definition.**—Conidiosporales in which reproduction takes place by aleuriospores

**Classification.**—The suborder contains two families which can be distinguished as follows —

A Conidiophores absent—*Aleurismaceæ*

B Conidiophores present—*Monotosporaceæ*

FAMILY ALEURISMACEÆ VUILLEMIN 1911

**Definition.**—*Aleuriosporineæ* without conidiophores

**Classification** —The family is divided into two tribes —

A Spores simple or appendiculate—*Aleurismææ*

B Spores bi- or multi cellular—*Blastotrichææ*

Only the first tribe is of interest to us

TRIBE ALEURISMÆÆ

This tribe may be classified as follows —

A *Hyphæ pale* —



III *Hyphæ elongate sporogenous apparatus well differentiated from the mycelium* —

- G sacchari* Spegazzini 1896 in the Argentine
- G microspora* Spegazzini 1891 in Brazil
- G khartoumensis* Chalmers and Archibald 1916, found in black maduromycosis
- G semoni* Chalmers and Archibald 1917

The various species may be recognized as follows —



Fig 598



Fig 599

FIG 598 AND 599 — *Glenspora khartoumensis* CHALMERS AND ARCHIBALD  
ALEURIOSPORES

- A Aleuriospores large usually measuring 10 or more microns in diameter —  
Parasitic on plants—(1) *Curtisia* (2) *Ramorum*
- B Aleuriospores medium measuring 6-8-9 or 11 microns—(3) *Sacchari*  
Parasitic on plants—(4) *Microspora*
- C Aleuriospores small usually measuring 5 or less microns in diameter —  
I Parasitic in man causing otomycosis and keratomycosis—  
(5) *Graphis*
- II Parasitic in r—  
(a) Growth at 30° abundant  
(b) Growth in exactly same conditions as in (a) Central series of small elevations from which radiate furrows cutting in black plateau Fringe very slightly marked—(7) *Khartoumensis* (in Africa)

***Glenspora graphis* Siebenmann 1889**

**Synonyms**—*Graphium penicillioides* Haller 1869 *Stemphylium  
illum graphis* Siebenmann 1889

aleuriospores large 10 or more microns in diameter  
Mycelium at first white later dark brownish The filaments are septated 2 to 3 μ in breadth ramified

masses of mycelium Siebenmann etc and in a case of keratomycosis by Morax and Pinoy

prickly appearance and consisting of erect straight filaments parallel to each other or at times interlacing. These filaments are approximately 2 microns in diameter, and carry laterally pseudoconidia of variable shape cylindrical pyriform or spherical.



FIG 595—MICROSCOPICAL APPEARANCE OF *Actadium castellanii* FUNGUS IN HANGING-DROP CULTURE FIVE DAYS OLD



FIG 596—MICROSCOPICAL APPEARANCE OF THE FUNGUS IN HANGING DROP CULTURE TWENTY FOUR HOURS OLD



FIG 597—MICROSCOPICAL APPEARANCE OF THE FUNGUS IN HANGING DROP CULTURE THREE DAYS OLD

S pseudoconidia a b c development of mycelial filaments from pseudoconidia ch chlamydospores

These pseudoconidia become detached and then develop by sprouting and mycelial filaments are formed. Certain filaments produce spherical chlamydospores arranged in small strings in the mycelium. These small chains are usually terminal the dimension (9, 597)

the colonies are white on glucose agar often amber colour. Old cultures may show a certain amount of pigmentation brown or black especially on potato

**Genus Glenospora** Berkeley and Curtis 1876

**Definition**—Aleurismae with pale and dark hyphae Aleurio

**History**—Found by Chalmers and Archibald in a case of black maduromycosis in Khartoum Anglo-Egyptian Sudan. It occurred in the sole of the foot of a native boy.

**Morphology**—All cultures show septate branched hyphæ varying in diameter from 2.8 to 4 microns and increasing in very old cultures when all the hyphæ are dark to 2.4 microns. At first the hyphæ are pale but when older they become dark being of a greenish black tinge. Thick walled clear or dark coloured. Chlamydo-spores (1.4 × 11.3 microns) are present and are especially marked in the black masses on the surface of potato infusions and nutrient gelatine. Aleurospores are to be found acropleur-

one  
well  
v

precipitate. On maltose agar the typical growth was in colour Ridgway's Standard Colour Dusky Drab and had a central elevation surrounded by a depression which separated it from a

eye Attempts to find

*African black maduromycosis*

*mycosis*

#### *Glenospora semoni* Chalmers and Archibald 1917

**Definition**—*Glenospora* closely resembling *G. khartoumensis* but differing markedly in cultural characters on maltose agar when grown under exactly similar conditions.

**Remarks**—This fungus was isolated by Senion from a case of black maduromycosis occurring in a native Indian soldier serving in France.

It is very like *G. khartoumensis* but differs markedly in cultural appearances as may be judged by a comparison of Figs 601 and 602 with Figs 603 and 604.

**Pathogenicity**—It causes a variety of *African black maduromycosis*.

#### Genus *Trichothecium* Link 1824

Fertile hyphæ are erect grouped together each terminating in an oval pear shaped or globular conidium. The only species so far observed in man is *Trichothecium roseum*.

**Type**—*Trichothecium roseum* (Persoon 1801) *emendatum* Link 1824

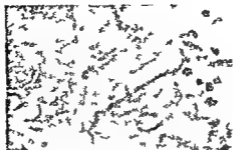


FIG 60—*Glenspora khartoumensis* CHALMERS AND ARCHIBALD IN MYCELIUM ON CRAIN

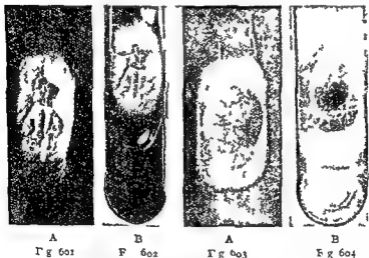


FIG 601 AND 602—*Glenspora khartoumensis* CHALMERS AND ARCHIBALD CULTURE ON CLEAR MALTOSE AGAR

A Twelve days B fourteen days

FIG 603 AND 604—*Glenspora khartoumensis* CHALMERS AND ARCHIBALD CULTURE ON CLEAR MALTOSE AGAR

A Twelve days B fourteen days

***Glenspora khartoumensis* Chalmers and Archibald 1916**

**Definition**—*Glenspora* with aleuriospores small 5 or less microns in diameter parasitic in man causing black maduromycosis and differing from *G. senensis* in cultural characters



And one may add —

10 *S bronchiale* Montagne 1844

There is however some difference of opinion with regard to some of these species, thus de Beurmann and Gougerot consider that the fourth and fifth are only varieties of the second

4 *S beurmanni* Matruchot and Ramond 1905 var *indicum* Castellani 1908

5 *S beurmanni* Matruchot and Ramond 1905 var *asteroides* Splendore 1908

Considering them all provisionally as separate species they may be recognized as follows —

A *Conidia* large (4 11 microns in greatest diameter) with well developed

B *Conidia* usually small with very short pedicle which may be absent —

I With radiating bodies *Conidia* very polymorphic round oval or bacilliiform varying from 4 8 microns in greatest diameter—*Asteroides*

II. Without radiating bodies —

(a) *Conidia* not numerous. Ferments lactose not saccharose—*Schenki*

(b) *Conidia* numerous —

1 Hyphæ 2 or more microns in diameter —

(A) Cultivated —

(1) Colonies dark from the first—*Gougeroti*

(2) Colonies whitish at first —

(1) Colonies finally black. Ferments saccharose not lactose—*Beurmanni*

(2) Colonies lightish brown—*Dori*

(3) Colonies whitish grey to black. Hyphæ wide 3 to 4 microns—*Indicum*

(B) Not cultivated. Hyphæ 5 to 7 microns in diameter—*Bronchiale*

2 Hyphæ less than 2 microns in diameter. Hyphæ 0.5 to 1 micron in diameter—*Jeanselmi*

potassium

*Sporotrichum schenki* Hektoen and Perkins 1900

Synonyms—*Sporothrix schenki* Hektoen and Perkins 1900, *Rhinocladium beurmanni* Verdun 1913

Discovered by Schenk in a case of gummatous lymphangitis in 1896 in North America. Easily grown on glucose maltose and other sugar media. Optimum temperature 30° to 38° C. Growth with an irregular surface generally of white colour but old cultures

*Trichothecium roseum* Persoon 1801

*Sporocentrum roseum* Persoon 1801 *Sporocentrum*

*roseum*

SUBORDER ~ SPOROTRICHINÆ VUILLEMIN 1910

1910

Genus *Sporotrichum* Link. 1809 emendavit Saccardo 1887

whether the human species belonged to this or to the other group

known to go

1887 (De la Roche)

05)

in diameter The colour is light brownish, never becomes black Does not grow on gelatine The mycelial filaments are very thin (0.5 to 1  $\mu$ ) with short mycelial segments occasionally dichotomous The fungus is not pathogenic, or very slightly so for rats and mice

*Sporotrichum gougeroti* Matruchot 1910

*Sporotrichum indicum* Castellani 1908

Found by Castellani in Ceylon It is doubtful whether it is a separate species or merely a variety of *Sp. beurmanni* The

*Sporotrichum jeanselmei* Brumpt and Langeron 1910

Was isolated by Jeanselme and P. Chevalier from a case of ' ' ' botanically by Brumpt

branches supporting clusters of spores The spores are oval or roundish ■ 5 to 3.5  $\mu$

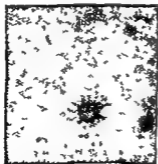


FIG 606—*Sporotrichum asteroides*

SPLENDORE IN THE TISSUES

Note radiate body

(From photographs by Dr Splendore)



FIG 607—*Sporotrichum aste*

*roides* SPLENDORE

Young culture

*Sporotrichum asteroides* Splendore 1908

Synonym — *Rhinocladus asteroides* Verdun 1913

Discovered by Splendore in South America Is characterized by

may present some brownish or black pigmentation. Glucose gelatine is slowly liquefied. Mycelial threads as a rule not very straight rather bent curved or undulating they are about  $2\ \mu$  in diameter. Conidia present in small numbers oval supported by a short sterigmata. Ferments lactose producing acidity but no gas has no action on saccharose.

**Pathogenicity**—It is the cause of Schenk's sporotrichosis found in North America. According to de Beurmann this fungus is very little or non pathogenic to rats and mice.

*Sporotrichum beurmanni* Matruchot and Ramond 1905

**Synonym**—*Rhinocladium beurmanni* Verdun 1913

Discovered by de Beurmann in France and completely investigated by Matruchot and Ramond. *C. ann. Bot. Soc. Lond. Paris*, etc.

glucose agar. The growth begins to appear between the fourth and the twelfth day. Optimum temperature  $22^{\circ}\text{C}$ . The growth may be whitish at first but soon becomes completely black or of a brownish chocolate-like colour. The surface is cerebriform. Glucose gelatine is slowly liquefied. Mycelial threads about  $2\ \mu$  in diameter rather straight. Conidia oval 5 to  $6\ \mu$  in length and  $3\ \mu$  in breadth supported by short sterigmata.



FIG 605 *Sporotrichum beurmanni* MATRUCHOT AND RAMOND (After Gougerot)

In contrast to *Sp. schenki* the conidia are extremely numerous.

Ferments saccharose producing acidity but no gas has no action on lactose.

**Pathogenicity**—Is the cause of by far the greatest number of cases of sporotrichosis in Europe (see p 2086). Is very pathogenic to rats and mice.

*Sporotrichum dori* de Beurmann and Gougerot 1908

Found by Dor in a case of gummatous sporotrichosis. In contrast to the typical *Sp. beurmanni* the growth on maltose and glucose agars is slower the colonies do not coalesce into a large mass but remain separate and small not exceeding  $1\frac{1}{2}$  millimetres.

**Acremonium potroni** Vuillemin 1911

Found by Potron and Noisette in a case of subcutaneous gum mata with fever somewhat resembling typhoid before the gum mata appeared. Easily grown on Sabouraud's agar colonies white then pinkish and later orange-yellow. Serum is liquefied. In cultures the mycelial filaments are septated, numerous conidiphores are present of a peculiar elongated type 15 to 20  $\mu$  in length. Conidia ovoid with a smooth surface 4 to 5  $\mu$  in length and 2 to 2.2  $\mu$  in breadth of pinkish colour. This fungus is pathogenic to guinea pigs.



FIG 608—*Acremonium potroni* VUILLEMIN  
(After Vuillemin)

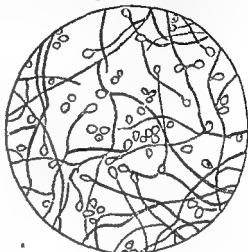


FIG 609—*Scedosporium apiospermum* SACCARDO  
(After Radaeli)

**Acremonium niveum** Boucher 1918

Very similar to *A. potroni* but colonies are generally white

**Genus Scedosporium** Saccardo 1911

**Definition**—Sporophorineae with unbranched decumbent conidiphores

**Type Species**—*Scedosporium apiospermum* (Saccardo 1911)

**Remarks**.—The other known species are *Scedosporium acremonoides* Harvey which does not concern us and *S. sclerotiale*

**Scedosporium apiospermum** (Saccardo 1911)

**Definition**—*Scedosporium* with mycelium at first white and later slightly brown bundles 3.5 mm broad in cultures. Mycelial hyphae creeping filiform. Conidiophores decumbent very slightly branched hyaline 2.5–3 microns with one spore oblong 1.4 × 5.6 × 5.7 rarely subround at first hyaline later dilute dirty rose yellow. *Scedosporium* causing white maduromycosis

**Remarks**—Easily grown the cultures are whitish and covered with duvet may become brownish when old

Mycelial tubes septated and of various size may reach 4 to 5  $\mu$

1		ies 4 to 12 $\mu$
1		ions radiate
1		the colonies
rice		an rats and

**Sporotrichum lesnel** (Vuillemin 1910)

Synonym — *Rhinocladium lesnei* Vuillemin 1910

Differs from all the other species of *Sporotrichum* by the elongated shape and large dimensions of the conidia which are 4 to 11  $\mu$  in length and 2.5 to 4  $\mu$  in breadth. Old cultures are of a dark sooty hue and black chlamydospores may be present.

*Sporotrichum councilmani* Wolbach Sisson and Meier 1917

SUBORDER 3 SPOROPHORINEÆ VUILLEMIN 1910

**Definition** — Conidiosporales reproducing by true conidia borne on conidiophores

**Remarks**.—A number of families and a large number of genera are included in this suborder but we are only concerned with four which may be separated from one another as follows —

A Conidiophores unbranched —

I Single hyaline or lightly coloured terminal spore—  
*Acremonium* Link 1809

B Conidiophores branched —

I Conidiophores erect—*Monosporium* Bonorden 1851

II Conidiophores decumbent—*Scedosporium* Saccardo 1911

*Monosporium* is only mentioned because *Scedosporium apiospermum* used to be *Monosporium apiospermum*

**Genus Acremonium** Link 1809

**Definition** — Sporophorineæ with creeping sterile hyphæ but little branched and carrying laterally simple unbranched conidiophores broad in the middle and gradually reduced towards the distal extremity terminating in a single hyaline or lightly coloured spore

Type Species — — — — —

which mostly  
Two species

**Scopulariopsis blochi** Matruchot 1911

**Synonyms**—*Mastigocladium blochi* Matruchot 1911 *Scopulariopsis blochi* Vuillemin 1911

Found by Bruno Bloch in a case of gummatous lymphangitis clinically very similar to an ordinary case of sporotrichosis. In cultures the mycelial threads are slender (0.5 to 1.5  $\mu$  in breadth) colourless septated very little ramified. Conidiophores 20 to 30  $\mu$  in length are tapering and from the pointed ends chains of conidia take origin. Conidia elongated ovoid 3 to 4  $\mu$  in length and 1.5 to 2  $\mu$  in breadth. In old cultures white creamy formations may be seen which may possibly be undeveloped perithecia.



FIG. 610—*Scopulariopsis blochi* MATRUCHOT (After Vuillemin)

**Scopulariopsis koningsii** Oudemans

**Synonyms**—*Monilia koningsii* Oudemans  
*Scopulariopsis rufulus* Baumer *S. koningsii* Vuillemin 1912

calls *S. verrucosus*

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in breadth Each conidiophore supports a terminal spore which is either ovoid 12 to 14  $\mu$  in length and 5 to 6  $\mu$  in breadth or roundish 6 to 7  $\mu$  in diameter No other kind of fructification is seen

### *Scedosporium sclerotiale* (Peperé 1914)

ex  
ga  
ca  
successful inoculations in the anterior chamber of the eye in guinea pigs

**Pathogenicity**—It was found in a case of black maduromycosis in a peasant aged thirty three years living at Domusnovas in the province of Cagliari in Sardinia

### SUBORDER 4 PHIALIDINEÆ VUILLEMIN 1910

**Definition**—Conidiosporales with conidia borne on phialides

**Remarks**—As already stated the species of *Aspergillus Sterigmatocystis* and *Penicillium* in which asci are unknown could be classified here but we will only consider *Scopulariopsis* which is closely related to *Penicillium*

### Genus *Scopulariopsis* Bannier 1907

**Synonym**—*Penicillium pro parte*

Bannier 1907

**Remarks**—In addition to those found in man *S. rubellus* Bannier

- A With white creamy formation in old cultures—*Blochii*
- B Without such formations in old cultures—*Koningsii*







# SECTION A

## FEVERS

### DIVISION I CAUSATION PROTOZOAL OR PROBABLY PROTOZOAL

#### SUBDIVISION A CARRIED BY MOSQUITOES

The Malarial Fevers  
The Tropical Hæmoglobinurias  
Yellow Fever  
Dengue and Allied Fevers

#### SUBDIVISION B CARRIED BY SAND FLIES

Pappataci Fever

#### SUBDIVISION C CARRIED BY TSETSE FLIES CONE NOSED BUGS AND UNKNOWN ARTHROPOIDS

The African Trypanosomiases  
The South American Trypanosomiasis  
The Kala Azars and Pseudo Kala Azars

#### SUBDIVISION D CARRIED BY LICE TICKS AND MITES

The Relapsing Fevers  
Typhus Fever  
The Spotted Fever of the Rocky Mountains  
Tsutsugamushi Fever

#### SUBDIVISION E CARRIED BY MAMMALS

Rat Bite and Cat Bite Fevers

### DIVISION II CAUSATION BACTERIAL OR PROBABLY BACTERIAL

The Enteroidæa Group of Fevers  
Plague  
Undulant Fever

### DIVISION III CAUSATION PHYSICAL OR PROBABLY PHYSICAL HEAT STROKE AND HEAT SYNCOPE

### DIVISION IV UNCLASSIFIED COSMOPOLITAN, AND WAR ZONE FEVERS

### DIVISION V DIFFERENTIAL DIAGNOSIS

**PART III**  
**THE DISEASES OF THE TROPICS**

- SECTION A FEVERS**
- SECTION B GENERAL DISEASES**
- SECTION C SYSTEMIC DISEASES**

*tertian fevers* and the *subtertian fevers*—in their typical and atypical acute phases. Having completed this we have still to consider the subject of chronic malaria, and we are then in a position to review the complications, sequelæ, diagnosis, and prognosis. Finally, the important matters of treatment and prophylaxis must be discussed.

It may be thought that it would be better to write three separate chapters detailing what is known considering the three types of fever separately and though scientifically this would be more accurate still, clinically it would not benefit, because it is in its atypical forms that malaria is mostly seen by the physician of to-day, and therefore clinically it is better to treat the malarial fevers together and not separately.

It is, however, necessary to preface the account of the disease with a very short note on the history in order that the reader may understand the salient features of the evolution of knowledge with regard to it.

**History.**—It is suggested that the references in the Charaka Samhitâ to fevers spread by mosquitoes refer to malaria and that this fever was

We thus see that very early in the history of medicine mosquitoes were associated with fever which was also associated with stagnant water and

## CHAPTER XL

# THE MALARIAL FEVERS

General account—Quartan fevers—Tertian fevers—Subtertian fevers—Pernicious malaria—Chronic malaria—Relapses—Reinfections—Complications—Sequelæ—Diagnosis—Prognosis—Treatment—Prophylaxis—References

### I. GENERAL ACCOUNT.

**Definition** The nature of the disease is that of a

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c

exciting cause of the subtertian fevers, which are carried from man to man by the agency of many species of anopheline mosquitoes

**Nomenclature.**—The name 'malaria' is derived from two Italian words, *mal aria*, meaning 'bad air,' and was intended originally to signify that the bad air arising from marshes was the cause of the fevers

**Synonyms.**—*English Names* Ague, paludism, marsh fever, remittent fever, intermittent fever, climatic fever, jungle fever, coast fever

*French Names* Fièvre palustre paludisme fièvres paludéennes fièvres des marais impaludisme, fièvres malariques, fièvres telluriques

*Italian Names* Paludismo, malaria, febbre intermittente, febbre palustre febbri malariche, febbri di stagione, febbri d'aria, infezione malarica

*German Names* Wechselfieber, Kaltes Fieber, Intermittens Sumpf fieber, Klimafieber, Marschfieber, Kaltes Fieber

**Local Names**—In all countries local names have been applied to the malarial fevers—for example, Roman fever Sierra fever, fever of Batavia, Kurunegala fever (Ceylon) Drumbul fever (Ceylon), Kamerun fever

**Seasonal Names**—As malarial fevers are more abundant in spring than in winter, and still more so in summer and autumn than in spring, it is natural that seasonal names should be applied to them—e.g., spring fevers, summer to autumn fevers autumn fevers

**Remarks.**—The malarial fevers cover such a wide field of clinical symptoms and pathological phenomena that it is necessary first of all to give a general account, which will comprise such subjects

With regard to this point the tendency at present is to believe that Schan

in nature

in this animal were mild but for several weeks typical parasites were found  
 5 Whether there are any malarial parasites of man which have so far not  
 been recognized

fourth day are malarial but this while correct as a general rule is open to  
 doubt in certain instances and in any case requires careful watch

**Ætiology** —The ætiology may be divided into —

- I The Exciting Causes.
- II The Predisposing Causes (p 1142)

I **EXCITING CAUSES** —Malarial fevers are produced by the para-  
 sites *Plasmodium malariae* Laveran 1881, *P. vivax* Grassi and Feletti  
 1890, and *Laverania malariae* Grassi and Feletti, 1890, because they  
 are always found in the blood or organs of persons suffering from  
 the disease, and can be injected into healthy persons, producing  
 in them typical fevers, the different stages of which correspond to  
 the stages of the life-cycle of the parasite. These parasites can be

mosquito was infected The classical experiment is the infection  
 of Sir Patrick Manson's son with tertian malaria by means of infected  
 anophelines sent from Rome to London

There are, therefore three factors necessary for the production  
 of malarial fever (1) the blood parasite, (2) the mosquito, (3) man

I **The Blood Parasite** —We have drawn attention to three para-  
 sites in connection with malaria and we believe these to be the only  
 three at present known to cause the fevers, but it is necessary to say  
 that this view is by no means universal Some authorities believe





a result, diarrhœa and urobilin in the urine. If the blood destruction is excessive, the liver is unable to convert the whole of the hæmoglobin liberated into bile, with the result that some may be left unaltered, and may produce hæmoglobinuria.

It appears, however, that an antitoxin is quickly formed of the nature of an anti auto-complement, which neutralizes this hæmo-

by the cells of the body, and anything which lessens its formation such as starvation gives the parasite a chance to grow and cause

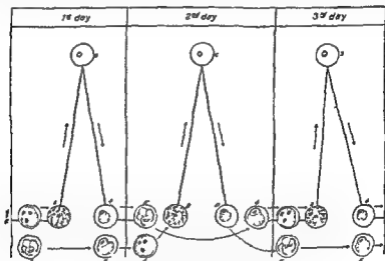


FIG 615.—DIAGRAM OF A TEMPERATURE CHART IN DOUBLE TERTIAN MALARIAL FEVER

The fever of the first and third days is due to one brood of parasites, and that of the second day to another brood

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of second  
of second

disease. Thus Casagrandi found that out of twenty one birds infected in 1904 ten were still infected in 1905. On semi starving two of these he produced relapses, while two others treated with antitoxin obtained from guinea pigs inoculated with pigeon's blood did not relapse.

It will thus be seen that a relapse may be due entirely to predisposing causes which lower the vitality of the body and prevent the production of sufficient quantities of the antitoxin.

Besides the difference pointed out above as to the place of sporula

sites have been given in Chapter XXI (p 594), and need not be

parasites they may either be killed and no infection result, or they

be said that some nine to twelve days are required for the development of a sufficient number of parasites to produce fever. Acton gives the following table of the length of the incubation period —

<i>Parasite</i>	<i>Maximum in Days</i>	<i>Minimum in Days</i>	<i>Average in Days</i>
Quartan	18	11	14
Tertian	21	6	11
Subtertian	14	2	6

During the incubation period, however there may be slight

#### Malaria

*The Fever*—The life history of the parasite has a definite re

The presence of *Anopheles* however does not indicate that there must be malaria in the locality an important point to which Celli was the first to call attention

England for example though it possesses three species of the *Anopheles*—viz *Anopheles maculipennis* Meigen 1818 *A. bifurcatus* Linnæus 1758 and *A. nigripes* St of which the two former certainly can carry malaria—is practically free from the disease Nuttall Cobbett and Strangeways Pigg believe that this condition has been arrived at by the reduction of the numbers of the mosquitoes by drainage

Since the war however a certain number of indigenous cases of benign tertian malaria have been reported

wi  
de

(Barbados for example as first noted by Low) Further it is highly probable that the endemic malaria of Mauritius and Réunion is due to the introduction of *Pyretophorus costalis* Loew 1866 as has been pointed out by Ross There is also the evidence that in places where successful anti-anopheline measures have been carried out as in Ismailia malarial fever has ceased to exist

In order that there may be plenty of these insects there must be a certain degree of warmth for as a rule they hibernate in the winter of the temperate zone coming out in the spring and increasing in numbers to reach a maximum in the warm days of autumn In the tropics of course the heat is present all the year round Heat alone however will not suffice for the mosquito for there must be water for the development of the larvæ and pupæ

occur until the summer or autumn This point has been carefully studied by Grassi Jancsó Hollander and others and the result of their experiments tends to show that temperature has most effect

will develop at a lower temperature than the other two while *P. vivax* will also develop at a low temperature but *L. malariae* requires a distinctly higher one

This may be the reason of the scarcity of *L. malariae* in the temperate zone except in the summer and autumn and its common

toxins and cause an attack of fever in its human victim every seventy two hours while in an infection with only one brood of

hours to complete its schizogony, and in single infections produces fever every forty eight hours—i. e. on the third day—and therefore a third time of fe

malarial fever—a benign and a malignant. The former caused by *P vivax* is generally called tertian malarial fever while the latter, caused by *L malaria* is called subtertian malarial fever, or malignant tertian

To add on to the a  
for the complet

or *L malaria* and does not require a special quotidian parasite. *Plasmodium fenu* Stephens 1913 appears in part to be related to *P vivax* and in part to *L malaria* while *P vivax minus* 1 of Emin, *P falciparum quotidianum* of Craig and Oswaldo Cruz's parasite require confirmation

Therefore there are only three parasites and three classes of fever to be considered—viz (1) *Plasmodium malaria* causing quartan malarial fever (2) *P vivax* causing tertian malarial fever

but remain near the place where they are developed. Occasionally they can be carried long distances by ships, trains, coaches, carts, etc., but this is the exception and not the rule. Winds do not appear to carry them far, as they generally take shelter from a high wind. The natural enemies of the Anophelinae are numerous, including all insectivorous animals, such as bats and birds, together with fish which eat the larvæ.

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1  
t  
malarial parasite and in this way it forms a reservoir of infection.  
3 Man—The female anopheline requires blood for the purpose of providing its eggs with sufficient nourishment and will therefore bite any vertebrate it may come across in order to obtain the same.

The malarial parasite hence until they are justified in assuming any other source. In the tropics the native population is undoubtedly the great source of infection of the mosquito; but in them do not protect themselves. In the native population the children are the greatest source of infection because as they lack immunity care must be taken sometimes swarming with gametocytes.

a given period depend upon—

- 1 The number of persons with gametocytes in the blood
- 2 The number of anophelines which have bitten these people and become infected
- 3 The number of infected Anophelines which live long enough to transmit the infection—i.e. at least a week.
- 4 The number of these surviving infected Anophelines which get the chance of biting man again.

occurrence in the tropics and possibly this is also the reason of the rarity of *P. malaria* in the low country of the tropics while its presence in the hills is common

Here may also lie the explanation of the universal distribution of *P. vivax* in both the tropics and temperate zone for Jancsó's researches show that it can develop through a wide range of temperatures

work has not been confirmed

fellow workers working on other portions of the estate will be but little affected. The explanation of this is not that emanations have

validity of these coolies and has given the germs already in their system a chance to develop

It has also long been thought that the mechanical opening of new ground by digging produced the disease but Ross has pointed out that in Mauritius the digging of earth for years caused no malaria

obtained in some other place where the mosquitoes are to be found

It may safely be concluded that as far as our present knowledge goes certain of the Anophelinae are the only carriers of malaria and upon this public prophylaxis must be based

A female mosquito apparently can live for at least a month (Ross) if not longer. This does not include such dormant periods of its life as the hibernation in the cold or aestivation in dry seasons when it may live for a long time in damp places

The eggs of the Anophelinae are laid only in natural collections of water supplied with water plants such as the back eddies of streams close under the banks which are especially good breeding places. The young imagines apparently do not usually travel

Then by the Poisson Pearson formula the percentage error will be—

$$e\% = \frac{200}{n} \sqrt{\frac{2x(n-x)}{n}} \sqrt{1 - \frac{n-1}{N-1}}$$

**Latent Malaria**—This term is employed to denote cases in which without any sign of illness malarial parasites can be found in the blood in small numbers. These cases form a reservoir of transmission to the anophelme. Crug states that out of 1267 cases in which malarial parasites were demonstrated in the blood 21 per cent were latent and the majority of these were found to be caused by the subtertian parasite.

**Congenital Infection**—The question of congenital malaria has been much debated but Dumolard and Viallet have recorded a

in the maternal blood and placenta. A similar case has been recently reported by Liger.

**II PREDISPOSING CAUSES**—These may be classified into—

1 Those which promote infection with the parasite  
2 Those which promote the increase of the parasite in man after its inoculation

**1 THOSE WHICH PROMOTE INFECTION WITH THE PARASITE**—The first of these is residence in an area which contains not merely persons with gametocytes in their blood but also mosquitoes capable of carrying the disease associated with an atmospheric temperature suitable for the development of the parasite in the mosquito.

marshy places and the wet season are important predisposing causes.

**AFTER ITS INOCULATION**—These predisposing causes are subdivisible into (a) racial (b) personal (c) meteorological.

(a) *Racial*—There is no doubt that the native races suffer less

present. Further there appears to be a tendency on the part of

*Investigation of an Endemic Region*—In investigating malaria in an endemic region it is necessary to find out—

1 The specific diagnosis of the parasite or parasites causing the malaria

5 The species of Anophelinae in the district and their breeding grounds

6 The species which carry the parasite

7 The species in which the parasite is found in nature

8 The number of Anophelines in the affected area

in 1.07 to 2 per cent of non malarial children living in London

Ross suggests that the term endemic index which was introduced by Stephens and Christophers to denote the percentage of persons carrying parasites in their blood should be extended to include not merely that factor but also the proportion of people

investigated Children are chosen because the adults have acquired

oss is sure to be higher  
the blood must be ex  
all the children whose spleens are not enlarged and the  
parasite-rate must be added to the spleen rate to obtain the true  
endemic index

$\frac{x}{100}$  = spleen rate

$e\%$  = percentage of error



AFRICA —The worst malarial region in the world is probably the West Coast of Africa, from Senegal to the Congo, but the whole of

the foot of the Himalayas, in Ceylon, and Borneo, while Arabia, Syria, the Straits Siam, and China are also malarial. Little is known about Siberia.

AUSTRALASIA AND POLYNESIA —Malaria occurs in the north of Western Australia, the Northern Territory, North Queensland, Torres Straits Islands, New Guinea, Finschhafen, the Solomon Islands, and the Bismarck Archipelago.

AMERICA —Malaria exists in Central America, the West Indies, with the exception of Barbados, the coast of the Mexican Gulf, the north of South America, including British Guiana, and the north of Brazil as far as Rio de Janeiro. Paraguay and Bolivia are infected, as are Peru and Chili, but the south part of South America is less infected. Many places in the United States are malarial, but Canada is not markedly infected, except about the northern shore of Lake Ontario, while Greenland is supposed to be free.

EUROPE —Great Britain and Norway are practically free, but most of the other countries have endemic foci, particularly Russia, Ita-

1

the

and

Sweden has also some endemic spots.

111.

there is generally a considerable amount of atmospheric moisture

necessary  
of alti

**Pathology** —In malaria the body is invaded by protozoal parasites which grow and increase at the expense of the red cells of the blood, and in doing so manufacture toxins of which we know two—viz., a pyrogenetic toxin and a hæmolysin.

Red blood cells are found in the whole of the circulatory organs, but are generally contained in arteries, veins, and capillaries. In two places, however—viz., the spleen and bone marrow—they come intimately into relationship with the parenchyma cells. Whatever function the spleen may in future be found to possess as regards the malarial parasites, it probably acts as a purifier to the blood which passes through it. Perhaps the bone marrow assists in such a function.

Parasites contained in red cells should, therefore, be able to pass

the body to manufacture protective substances, which keep the

(2) starvation or overfeeding; (3) the onset of another acute disease, (4) the presence of some chronic ailment, which may often be but slight

(c) *Meteorological*—We have already drawn attention to the relationship between the temperature of the external air and the development of the parasites in the mosquito. It now remains to point out that a similar relationship exists between that condition and the development of the parasite in the human being.

Ross is of the opinion, not merely from observations upon man infected with malaria, but also upon birds infected with *Halteridium*, that high air temperatures are favourable to the increase of the malarial parasites in man. High air temperatures are therefore a cause of the relapses met with so frequently in the hot dry season of the tropics.

The reverse is also true, hence the benefit of sanatoria at high altitudes in the tropics, and also of sending a fever-stricken patient to cooler climates, provided the change from the hot to the cold

quitoes, together with the presence of human beings with numbers

those organs. After a series of attacks the blood capillaries and lymph spaces in the liver and spleen remain permanently dilated and separated by only a slight amount of damaged parenchymal tissue. Later, regeneration of the parenchyma takes place, but the organ will remain permanently altered, even though all pigmentation may have disappeared.

In the case of  
be caused to the  
by the parasites

may damage the latter organ

There are two main conditions the pathology of which must be explained—viz, acute and chronic malaria. In acute malaria there are the effects produced by each of the three parasites, of which the subtertian is liable to seriously damage important organs.

Chronic malaria should also be described according to the three types, but there is at present lack of material to evolve such a description. Chronic malaria may however, pass to an advanced condition called 'malarial cachexia,' which shows itself in three ways—as (1) a rare acute cachexia, (2) a more common chronic cachexia, (3) cachexia with amyloidosis.

In addition, there are the pathological features of latent malaria and the relapses.

Before, however, proceeding to describe the actual morbid anatomy of these conditions, a few words must be said upon what we know of the chemical pathology of the disease and on the blood changes which take place.

**Chemical Pathology.**—The pyretogenous toxin has already been mentioned among the poisons of animal origin and, though long suspected of being present, its actual occurrence was first proved by Rosenau and his collaborators. Probably it is the poison which acts deleteriously upon the tissues of the organs and causes metabolic changes, but this is only a matter of conjecture.

We know that the heat output in the cold stage of the attack is markedly diminished—a condition met with in many fevers—but the chemistry of the metabolic changes is but little known. During

shown by the large increase in solids. The colour is dark, and the acidity of the urine is increased, as in most fevers. Nitrogen is excreted in excess, which is largely due to the increase of urea. Chlorides, sulphates and bases, especially potassium, are all increased in quantity. Phosphates, however, are diminished during

all over the body and should be found equally distributed no matter

the red cell by the quartan parasites is not severe enough to cause them to be caught in the capillaries. Therefore the whole life history of the quartan is spent in the circulation and sporulating forms can be readily seen in finger blood.

Tertian parasites on the other hand seriously affect the red cells causing swelling, degeneration and decolorization. The trophozoite and schizont stages are easily seen in the peripheral circulation. Still the sporulating forms are more common in the spleen which may be looked upon as having filtered them off from the blood which passes through it.

The subtertian parasites act deleteriously on the corpuscles making them smaller and darker. They rarely appear in the peripheral circulation in the sporulating condition while they abound in the spleen and internal organs. On examining the organs post mortem it will be found that the schizont and sporulating forms

black spots next to the ...

leucocytes, macrophages and the endothelial cells of capillaries therefore in acute malaria it will be distributed evenly through the organ affected. The pigment is later conveyed from the blood

as the ... deposited in the parenchyma cells of the organs—

for the arrectores pilorum are also affected producing goose-skin. *Laverania malariae* however, because it sporulates internally and not in the peripheral blood causes chills more rarely than the other two.

has a restraining power against increase of the parasites.

The only other remarks we can offer on the chemical pathology are limited to the nature of the pigments hæmozoïn and hæmosiderin.

glc

an

but not in water alcohol chloroform ether or acids. It contains iron but in the form of the Berlin blue reactive tissue cells used in some altered

malarial parasites it is peculiar to the diseases caused by them. Brown considers that it is formed by the action of a proteolytic enzyme from the parasite acting upon the hæmoglobin of the erythrocyte and that therefore it is formed from hæmatin and

cytes after any great destruction of blood cells. In malaria it is undoubtedly due to the action of hæmolysins destroying the red

numbers

the actual attack but increased as it is passing off and are considerably increased in the intermission. Phosphoric acid is therefore retained in the body during the attack.

As would be expected iron is excreted in increased quantity probably due to hæmoglobin and hæmosiderin but this increase does not really appear until after the actual attack is over and then continues for some days.

As before remarked the urine may contain a considerable quantity of urobilin and the indigo blue may be also increased.

THE

URINE

During convalescence the most marked features are the polyuria with low specific gravity which in subtertian fevers may be so marked as to alarm the patient while in quartan and tertian it may be so slight as to escape attention. There is also increase of chlorides and potassium salts excreted.

Urriola states that in the urine in malarial cases present four types of pigment granules may be found (1) very fine granules (2) larger granules in groups (3) large masses (4) granules in leucocytes and hyaline casts. It is however difficult to exclude extraneous matters.

As regards the fæces the most noticeable feature is the increase in the excretion of bile and iron both of which are related to the

odour and contains substances very toxic to rabbits.

The above chemical features point to the fact that the toxins of

*First Type*—This form of anæmia comes on after attacks of ordinary acute malaria and is characterized by well marked diminution in the erythrocytes the presence of normoblasts diminution of the colour index and leucopenia associated with relative mononuclear increase.

*Second Type*—The exceedingly bad It red cells presence of megaloblasts with leucopenia and relative mononuclear increase

*Third Type*—This is rapidly fatal and has similar characters to

Wassermann reaction is in our experience generally negative

**Morbid Anatomy**—The morbid anatomy of malaria has been most carefully studied in recent years by Bignami in Italy and Ewing in America It may be considered under the following headings —

#### A ACUTE MALARIA

- 1 Lesions caused by *Plasmodium malariae*
- 2 Lesions caused by *Plasmodium vivax*
- 3 Lesions caused by *Laverania malariae*

#### B CHRONIC MALARIA

- 1 Lesions caused by *Plasmodium vivax*
- 2 Lesions caused by *Laverania malariae*
- 3 Malarial cachexia
- 4 Latent malaria

**A MORBID ANATOMY OF ACUTE MALARIA—1 LESIONS PRODUCED BY *Plasmodium malariae***—*Plasmodium malariae* goes through the entire process of schizogony in the circulating blood and hence is evenly distributed all over the body and therefore does not especially accumulate in any one organ Marchiafava and Bignami mention that they have made two autopsies one on a case of acute quartan malaria in which the patient died of nephritis and the other in a case of the same fever in which the patient died of spinal disease

The visceral lesions are Spleen enlarged not softened nor very melanotic liver and bone marrow not markedly melanotic parasites in the spleen and in the blood but not in the brain

Leishman has mentioned that he has received films from the peripheral blood and spleen of a fatal case in both of which the parasites were very numerous but he was not in a position to state whether the patient died of malaria or not If *P malariae* is to produce severe symptoms it would appear necessary for it to exist in very large numbers.

called 'latent malaria' and can be easily converted into active malaria by any cause which depresses the vitality of the body

**ERYTHROCYTES**—In quartan malaria the corpuscle containing the parasite is a little smaller than a normal corpuscle, and if anything, more darkly coloured. In tertian malaria it is swollen and more lightly coloured, and on treatment with Leishman's stain exhibits fine red granules (Schuffner's dots), which are to be looked upon as a sign of degeneration of the corpuscle. In subtertian malaria the corpuscles when stained in the same way may exhibit Maurer's dots or clefts, which appear as large, irregular red

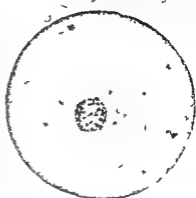


FIG 616—BLOOD FILM SHOWING SCHIZONT OF *Plasmodium vivax*  
(From a microphotograph by J J Bell)

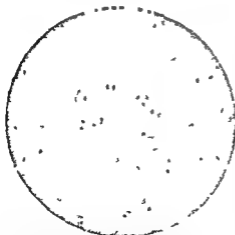


FIG 617—BLOOD FILM SHOWING HEAVY INFECTION WITH *Laveran's malaria*  
(From a microphotograph by J J Bell)

brass. These are corpuscles which have undergone some form of necrosis, probably due to the hæmolysin, though it has been thought that they were infected corpuscles in which the parasite had died as a result of the necrosis.

Partial decolorization of the erythrocytes has been recorded in subtertian infections and is especially well marked in those containing crescent bodies. Bignami thinks that in the subtertian fever the red corpuscles, which are infected with the

parasite, have a diminished elasticity, and therefore are not so capable of circulating and cling to the walls of small capillaries.



leptomeninges which may be oedematous and thickened (2) brownish or even blackish pigmentation of the cortex and (3) punctiform hæmorrhages in the white matter under the cortex or elsewhere. The spinal cord exhibits changes similar to those in the brain while the retina shows numerous hæmorrhages.

*Microscopical Examination*—After death parasites may be found in the blood of the heart spleen bone marrow and at times in the capillaries of the brain intestines pancreas etc.

The parasite however is much shrunken and the typical forms seen during life are not distinct after death. Thus the ring form shrinks and becomes a rounded disc with the chromatin particle situated at the periphery. The fully developed schizont is more typical the merozoites being arranged in a ring around the central pigment block. If the post mortem is made quickly this shrinking is not noted. The mononuclear leucocytes will be noted to have pigment granules while the polymorphonuclears may show phagocytosis. In films from the internal organs macrophages with parasites and red cells may be seen.

In the heart there may be a few parasites but very rarely the capillaries will be found filled with red cells containing numerous parasites and the heart muscle laden with hæmosiderin as has been described in a pernicious cardiac

cells often laden with pigment. These capillaries are filled with blood cells

be present

The capillaries of the abdominal fat are often full of red cells containing parasites. The bone marrow is chocolate coloured in the small bones and brownish red in the long bones. Often it is soft and diffident and contains sporulating parasites and in particular crescents (gametocytes) which are

necrotic but the submucosa and deeper layers escape injury and their blood vessels contain nearly normal cells

the  
s of  
are

2 LESIONS PRODUCED BY *Plasmodium vivax*—It is rare for

The principal features are the pigmentation of the bone marrow liver and spleen which last is also enlarged. The blood and spleen show large numbers of *P. vivax*. The kidneys and colon are inflamed and the endothelial cells of the brain are swollen and contain pigment.

3 LESIONS PRODUCED BY *Laverania malaris*—We have already insisted several times upon the fact that *L. malaris* differs from the other malarial parasites in sporulating in the organs

the heart or the pancreas producing marked signs of disease therein. Therefore the conditions of the organs vary with the localization of the parasite.

examination

The anæmia is marked and there is fluid about the ankles and in the abdominal cavity. The spleen is enormously enlarged as is also the liver while the bone-marrow is yellow sclerotic or gelatinous. Parasites may be found or they may be absent.

A special form of cachexia is that in which amyloid changes are

can exist in the spleen of persons who show no sign of fever or malarial cachexia. These parasites can go through their life-cycle in that organ and in the case of *L. malariae* in the liver also but it would appear that they are restrained from invading the circulation by the action of some antitoxin and therefore do not increase to such numbers as to cause toxic symptoms.

It is obvious from the above that if the restraining influences which conduce to the condition of latent malaria are removed an attack of malaria will follow or if there has been a previous attack a relapse will take place.

Observers have always had a difficulty in admitting that the ordinary form of the parasite could be latent and cause a relapse

thus starting a cycle of schizogony anew and causing fever. Craig and other observers insist upon conjugation causing a rejuvenescence of the parasite and a relapse of the fever.

**Classification**—As there are three parasites—*Plasmodium malariae*, *P. vivax* and *Laverania malariae*—there are therefore three clinical entities—quartan malarial fever, tertian malarial fever and subtertian malarial fever—due to these parasites.

almost entirely  
is especially  
features to

*atypical subtertian fevers*, and is also the basis of those serious symptoms which have for many years been alluded to as the perniciousness of this type of fever. The nature of these pernicious symptoms will depend upon whether the parasite is principally localized in—(1) the cerebro spinal nervous system, (2) the gastrointestinal mucosa, (3) the pancreas, (4) the heart, (5) the lungs, (6) the liver, etc.

We will give clinical descriptions of the different quartan, tertian and subtertian fevers.

The brain in pernicious cerebral fevers has its capillaries filled with spore

**B MORBID ANATOMY OF CHRONIC MALARIA**—The lesions of chronic malaria fall principally upon the spleen the liver and the bone marrow. There are no records of post mortems on persons suffering from chronic malaria due to *Plasmodium malarie*.

**1 LESIONS DUE TO *Plasmodium vivax***—The best recorded case of this infection is that given by Ewing of a man who suffered from the disease for about a year and died from endocarditis about three months after the last attack.

mented showing a few endothelial cells with black pigment

**2 CHRONIC MALARIA WITHOUT DEFINITION OF THE PARASITE**—These lesions are generally due to *Lacerantia malarie* and as stated above affect the spleen liver and bone-marrow.

The spleen is always enlarged often considerably and is firm in consistence

of which are thickened

**3 MALARIAL CACHEXIA**—This may be acute when it develops after a few attacks of fever but more commonly it comes on as a sequela to chronic malaria.

when anopheline mosquitoes of undetermined species were allowed to bite men in India from the 9th to the 21st of January with the result that one developed a temperature of  $38^{\circ}\text{C}$  on the 5th of February but parasites were not found and the other cases were negative

**Remarks**—The clinical description may be divided into the febrile attack and the apyrexial interval

**THE FEBRILE ATTACK**—Generally there are prodromata before an attack of quartan fever. Some few hours previously the patient may complain of giddiness, weakness, malaise, headache or even nausea and vomiting. If the blood is examined during the occurrence of these symptoms the parasites will be seen to be schizonts and the commencing formation of merozoites may also be noted.

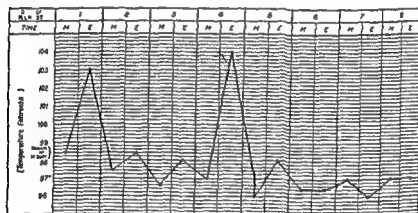


FIG 619—TEMPERATURE CHART OF A CASE OF SIMPLE QUARTAN MALARIAL FEVER IN THE GENERAL HOSPITAL COLONBO

In a short time the definite attack begins. It may roughly be divided into three stages: (1) The cold stage, (2) the hot stage, (3) the sweating stage.

**I The Cold Stage**—The patient begins to feel cold either in the legs, arms or back. This sensation increases until actual shivering sets in. In quartan fever the rigors are well marked and characteristic and the patient may shiver until he shakes the bed, the teeth may chatter, the lips become blue, the arms and legs cold and goose skin may be present. If the blood is examined in this stage some of the parasites may be seen fully sporulated or only young parasites may be found.

During this cold stage which is the most uncomfortable of the three stages the internal temperature is rising rapidly and the internal organs must be somewhat congested during the chill for there are symptoms of intense headache, visual disturbance, vomiting and at times diarrhoea. The temperature which rises

## II THE QUARTAN FEVERS

*Quartan malarial fever* depends for its symptoms and course upon the life history of *Plasmodium malariae* introduced into the blood of man by an anopheline mosquito. Its clinical course will depend entirely upon whether the parasites are of approximately the same age or whether they have been introduced into the body on different days.

If only parasites of approximately one age exist in the blood a typical quartan malarial fever ensues with an interval of seventy-two hours (the length of time which a merozoite takes to become the fully developed schizont). Such a fever is called *quartana simplex* or simple quartan fever.

If however the parasites were introduced on three successive days the fever may be daily—that is to say may be a quotidian.

There are therefore several types of quartan fevers—viz —

- A *Acute quartan malaria* —
- 1 Simple quartan fever
  - 2 Double quartan fever
  - 3 Triple or quotidian quartan fever
  - 4 Irregular subcontinuous quartan fevers
  - 5 Mixed infections
- B *Chronic quartan malaria*

### Simple Quartan Fever

**Definition**—*Simple quartan malaria* is characterized by attacks of fever recurring every seventy-two hours and separated by apyrexial intervals which occupy the time required by *Plasmodium malariae* to pass from the merozoite to the fully developed schizont.

**Incubation**—This has not been determined with any degree of certainty. It is without doubt longer than either tertian or subtertian. Cells by experiment came to the conclusion that it might be very long—two months or more.

know is the very doubtful one recorded by Buchanan in 1901

### Double Quartan Fever

¶ In this form there is an attack of fever on two successive days

stated this may be due to two groups of parasites inoculated on separate days. But sometimes quartana simplex may become quartana duplex, and this is explained by the fact that there may be a double infection but that while there are many of one brood of parasites and hence fever the other brood may be so few at first that they require time to develop to such numbers as are necessary for the production of fever. Consequently quartana duplex may at first show itself by a very slight rise in the temperature on the second day which increases gradually till equal to that produced by the stronger infection.

### Triple Quartan Fever

This is a quotidian or daily fever produced by three broods of quartan parasites coming to maturity on three successive days and can only be diagnosed by an examination of the blood. The three attacks may be similar and may begin at the same hour or they may vary in severity and begin at different times. In quartana triplex sometimes an attack may be subintra-

ture

A simple or a double quartan may become a triple quartan in the manner described above for the origin of a double from a simple fever. On the other hand it may start as quartana triplex and become a duplex and finally a simplex. This may be due to the weakening of certain groups of parasites. Sometimes a triplex may directly become a simplex from the linking together of two other groups of parasites at the same time.

### Irregular Subcontinuous Quartan Fevers

Quartan parasites are believed not to cause continuous fever but very rarely they may cause subcontinuous or remittent fever

fever

### Mixed Infections

Mixed infections may occur with either of the other two parasites—viz *P. vivax* or *L. malarie*—and an intermittent irregular fever be produced only to be diagnosed by the microscope

### Chronic Quartan Malaria

See Chronic Malaria p 1182

### III. THE TERTIAN FEVERS.

*Tertian malarial fever* depends for its symptoms and course upon the life history of the parasite.

days  
 If only parasites of approximately one age exist in the blood, the fever is quotidian.

duced on two different days, and are therefore of different ages, the patient will develop fever every day. Such a fever would be quotidian in type, and would be called 'tertiana duplex,' or double tertian fever.

If many broods of parasites are present, the fever becomes sub-continuous and irregular.

There are, therefore, several types of tertian fevers—viz —

A . . . . .

#### B *Chronic tertian malaria*

##### Simple Tertian Fever.

**Definition**—*Simple tertian malaria* is characterized by attacks of fever recurring every forty-eight hours and separated by aperiodical intervals which occupy the time required by *Plasmodium vivax* to pass from the merozoite to the fully developed schizont.

**Incubation**.—The natural incubation period is believed to be from eight days upwards. The period of incubation is aperiodic.

other hand, these may be most characteristic. When present



Remarks.—Only the bilious remittent will be considered here, as malarial hæmoglobinuria will be treated in the next chapter on Tropical Hæmoglobinurias, and the atypical subtertian fevers will be described directly after bilious remittent fever

### 5 Billous Remittent Fever.

blc

ha

Zone, and it is said to occur in all highly malarious districts.

The attack begins as an ordinary remittent fever, but is asso-

a few days' illness the symptoms may gradually subside, or, with or m.

of

by

### 6 Mixed Infections.

Mixed infections of *L. malariae* with the other malarial parasites are not uncommon and lead to a type of quotidian fever which can only be diagnosed accurately by an examination of the blood and a differentiation of the parasites concerned

The blood may show only forms belonging to *P. vivax* at times and only forms belonging to *L. malariae* at other times

## B ATYPICAL SUBTERTIAN FEVERS

The causation of this group of subtertian infections is due to the fact that *Laverania malariae* undergoes schizogony in the internal

laden with pigment, and by the pigment and merozoites set free

or malarial mimicry

These various syndromes may for purposes of description be arranged as follows —

- I Subtertian syndromes without localization
- II Subtertian syndromes with localization

I SUBTERTIAN SYNDROMES WITHOUT LOCALIZATION

Though it is true that one and the same case may show fever on

A NON-LOCALIZED SUBTERTIAN SYNDROMES WITHOUT MARKED FEVER ON FIRST EXAMINATION

- 1 Hæmorrhagic non febrile type
- 2 Anæmic type
- 3 Mental types
- 4 Algide type
- 5 Pseudo alcoholic type

*Hæmorrhagic Non-Febrile Type* —The patients are pale, very weak, and languid, and complain of pains in the loins and limbs. In most cases the whole body is covered with petechiæ, and tense  
 . . . of blood may be  
 hagic  
 ' take  
 idder

after repeated examinations while the usual signs of *perniciosa* *anæmia*—e.g., poikilocytosis nucleated red blood corpuscles, high colour index, relative increase of small mononuclears in place of the increase of the large mononuclears which one expects to see in malaria may be present

. . . most malignant  
 pain

Quinine acts as the diagnostic and therapeutic agent.

*Leukæmia* —Certain authors believe that malaria can cause leukæmia but this appears to us to be more of the nature of a complication

*Mental Types* —The patient is melancholic or apparently demented, or more rarely acutely maniacal and violent, with usually a normal, subnormal, or but slightly raised temperature. The spleen may or may not be enlarged, but a careful examination of the blood

parasites while quinine therapy effects a disappearance of the symptoms

*Algide Type* —The patient is as a rule first seen in a condition of such extreme collapse as to make the practitioner suspicious of cholera. The nose is sharp, the cheeks sunken, the lips and ex-

scious and be able to answer questions and to complain in a weak voice of severe thirst. This is a very serious and fatal form of

may or may not take a certain amount of alcohol. Suddenly during or after a dinner or at a public performance he tumbles off

the subtertian type. At a post mortem these parasites will be

will be found to be seriously ill and may even be dying. Blood examination generally but not always reveals malarial parasites. Generally these are serious infections and the prognosis is grave.

#### B NON LOCALIZED SUBTERTIAN SYNDROMES WITH MARKED FEVER ON FIRST EXAMINATION

The non localized subtertian syndromes with marked fever on first examination may be divided into —

- I
- II
- 3

#### Subtertian Hyperpyrexial Fever

temperatures  
continuous  
105° F  
hardly

be believed—*eg*, in two of our cases the temperature exceeded  $108^{\circ} \text{F}$ . These cases have a very serious prognosis, but recoveries are not unknown.

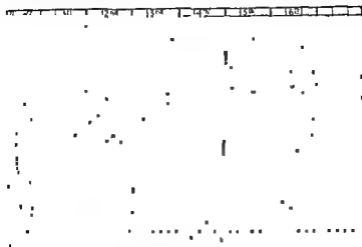


FIG 623.—TEMPERATURE CHART OF A CASE OF HYPERPYREXIAL SUBTERTIAN MALARIA.

#### Syndromes resembling a Specific Fever.

The syndromes resembling a specific fever may be divided into types as follows—

- 1 Typhoid like type
- 2 Malta fever like type
- 3 Typhus like type
- 4 Cerebro spinal like type
- 5 Yellow fever like type
- 6 Weil's disease like type
- 7 Scarlet fever-like type

*Typhoid like Type*—This fever resembles enteric fever, as may be seen from the chart. The onset is slow, and the fever is present, as in typhoid. The temperature is not large and the patient does not respond to quinine. The duration is long, and the prognosis is fatal.

*Malta Fever like Type*—This is very rare and resembles Malta fever. The onset is slow and the fever is present, as in Malta fever. The temperature is not large and the patient does not respond to quinine. The duration is long and the prognosis is fatal.

bacteriological tests for *Micrococcus melitensis* and its allies are absent. Finally quinine effects a cure.

*Typhus like Type*—The cases exactly resemble typhus fever but malarial parasites are often present in the blood and the disease

number and character while meningococci are absent and the blood shows malarial parasites. The spleen may or may not be enlarged.

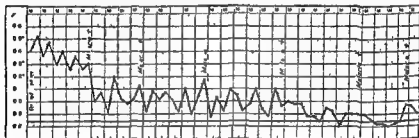


FIG 624—ATYPICAL SUBTERTIAN MALARIA SIMULATING TYPHOID FEVER  
Hæmocultures and serological reactions for germs of the Enterococci group of fevers were negative

*Sleeping Sickness like Type*—This which is rare is characterized by low fever slight trembling of the hands and tongue and progressive general weakness drowsiness and occasional convulsions. The lymph glands in the neck occur in regions where sleeping parasites may be hard to find affection.

*Yellow Fever like Type*—This is characterized by fever without

while the patient feels better but the temperature rises again jaundice appears the pulse slows to about 60-70 per minute dark brown vomit appears containing red blood corpuscles. The symptoms may get worse the jaundice may deepen and death ensue. Subtertian parasites may be present in abundance in the blood and quinine may be ineffective unless given in massive doses.

*Weil's Disease like Type*—Cases like Weil's disease with cutaneous malarial and may end fatally also be simulated by malaria.

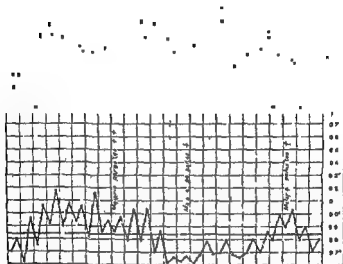


FIG 625—ATYPICAL MALARIA SIMULATING MALTA FEVER

Hemocultures and serological reactions for *Micrococcus melitensis* and *M. parameitensis* were negative

*Scarlet Fever like Type*—Scarlatiform pernicious fever is characterized by a diffuse scarlatiform rash all over the body with desquamation of the horny layer and erythema of the fauces, and may lead to a typhoidal state in which the patient dies

#### Syndromes not resembling Specific Fevers

The subtertian syndromes not resembling specific fevers are —

- 1 Hemorrhagic febrile type
- 2 Hydrophobia like type
- 3 Kussmaul coma like type
- 4 Diaphoretic like type
- 5 Comatose type
- 6 Delirious type
- 7 Tetanus like type
- 8 Convulsive type

skin and mucosæ of the nose, bronchi, intestines, stomach, and generative organs, during the attack, but not during the intermission.

This condition rapidly produces acute grave anæmia, with thready pulse, coma, delirium, convulsions, and death, or may become the milder type, in which fever is not a marked symptom (*vide p 1169*)

*Hydrophobia-like Type*—Signs of hydrophobia associated with fever, a large spleen, and malarial parasites in the blood, call for

sionally after the exhibition of a small dose of quinine, parasites may be found in the blood

*Diaphoretic Type*—In this type the sweating of the third stage of an attack is so exceedingly copious that not merely is the bed saturated, but a pool may even form on the floor. Such excessive ex-

latter, there may be weakness sleepiness, headache, disturbance of vision, stupor, or delirium, which ends in coma. But more often the patient is brought to the hospital quite comatose. He lies flat upon his back, with usually no paralysis and no alteration in the reflexes. The pupils may be contracted and give the idea of opium-poisoning, while the patient cannot be roused to answer questions, but will only frown or groan.

Hæmorrhages may be found on the skin and in the retina. The urine, which may have casts and a little albumen, is usually passed involuntarily, as are the motions. The heart is dilated, and the pulse, at first slow, soon becomes quick, and towards the fatal termination very quick and thready. Respiration may be quiet or noisy. If death is to take place, the patient becomes colder and

gradually recovers consciousness. The speech is at times most peculiar, being scanning in character.

The coma may be present one day, but the next day the patient l temperature and a slower, the temperature rises, and c is, as a rule, fatal. The fever may be remittent or intermittent. In the latter case it

*Delirious Type*—In this form delirium is the most marked feature and in a short time is followed by exhaustion coma and death. Such cases are apt to come on suddenly and to be mistaken for drunkenness sunstroke or mania. The fever is usually very high. Recovery is supposed to occur but we have never seen these cases end otherwise than fatally.

*Tetanic Type*—Patients delirious from malaria may show trismus contraction of the limbs opisthotonos retraction of the abdomen and conjugate deviation of the eyes. The contractions of the muscles may relax and increase as in tetanus and there may be priapism. The attack may resolve or end in death with high temperature.

*Convulsive Type*—Children during an attack of fever may develop convulsions followed by stupor or even coma and death.

## II SUBTERTIAN SYNDROMES WITH LOCALIZATION

The subtertian syndromes with localization may be classified into —

- I Syndromes with nervous system localization
- II Syndromes with digestive system localization
- III Syndromes with respiratory system localization
- IV Syndromes with circulatory system localization
- V Syndromes with ductless glands localization
- VI Syndromes with urogenital localization
- VII Syndromes with cutaneous localization
- VIII Syndromes with special sense localization

### I WITH NERVOUS SYSTEM LOCALIZATION

The atypical subtertian malarias with nervous system symptoms may be classified as follows —

- 1 Meningitic type
- 2 Hemiplegic type
- 3 Monoplegic type
- 4 Myelitic type
- 5 Ataxic type
- 6 Disseminated sclerotic type.
- 7 Bulbar type
- 8 Cerebellar type
- 9 Cerebral type
- 10 Polyneuritic type.
- 11 Korsakoff type
- 12 Aphasic type

*Meningitic Type*—Fever with signs of meningitis may be found in young persons and children. The symptoms are vomiting headache retraction of the head and rigidity of the neck convulsions hyperesthesia going on to coma and death. There may be hypertension and slight lymphocytosis of the cerebrospinal fluid.



*Hemiplegic Type*—The patient may be suddenly taken ill with fever, and develop a typical hemiplegic attack.

first there is no fever, but this may develop later. Such patients generally recover on quinine treatment, or, indeed they do so spontaneously.

*Disseminated Sclerotic Type*—There is scanning speech, intentional tremor, nystagmus, spastic gait, and increased reflexes, all of which yield to quinine therapy.

*Bulbar Type*—A fever with bulbar symptoms—that is to say, difficulty of speech and deglutition, with facial paralysis, or with a weakening of the muscles of the face and also frequently of the hands and feet. The condition yields to quinine therapy, but the fever is often absent from the beginning.

Other types are ataxia, the explanation of severe headache and almost complete loss of vision. Vomiting is frequent, often there is no fever and the spleen and liver are not enlarged. The blood may show parasites, and quinine therapy cures the condition, which may be suspected to be cerebellar tumour or abscess.

quinine cures the condition.

If there is no fever cerebral tumour may be suspected, but the blood examination reveals the true condition.

*Polynuritic Type*—This type closely simulates wet beriberi, because the patient is œdematous, with the characteristic gait and loss of knee jerks. There is neither fever nor enlargement of the liver or spleen, but the blood contains subtertian parasites, and the condition is cured by quinine therapy.

the mental symptoms, among which the most important is the loss of memory for recent events, all of which disappear under the influence of treatment by quinine. Subtertian parasites may or may not be present, but the spleen is not enlarged and the liver does not develop with or

II. WITH 'DIGESTIVE SYSTEM' LOCALIZATION

The subtertian malarías with digestive system symptoms may be classified as follows —

- 1 Pseudo cholera type
- 2 Pseudo-dysentery type
- 3 Pseudo appendicitis type
- 4 Pseudo peritonitic type
- 5 Pseudo liver abscess type
- 6 Pseudo cholecystitis type
- 7 Pseudo-cirrhosis type
- 8 Hæmorrhagic pancreatitis type.
- 9 Gastritis type.

*Pseudo-Cholera Type* — This is merely a great exacerbation of the ordinary gastro-intestinal symptoms often met with in malarial fever. There is vomiting abdominal pain and severe diarrhœa with motions typical of cholera — *i. e.*, rice water motions. A microscopical examination of the dejecta may show a few leucocytes in stages of degeneration. The spleen may be palpable, and an examination of the blood will show subtertian parasites while a bacteriological examination shows absence of cholera and of paracholera vibrios

sensible to the end. If the patient is to recover the algidity diminishes, the diarrhœa ceases and after a long sleep he awakens

with little or no mucus. Fever may be high with great distress and prostration and a small rapid pulse but at times the temperature may be nearly normal. The spleen may be slightly enlarged there may be history of previous malaria and there may or may not be

blood, and the condition yields to quinine therapy by proper means.

*versâ*

*Pseudo-Cholelithic Type*—There is severe pain, shooting into

seems an attack of cholecystitis due to gall stones, is cured by quinine.

cures the condition

*Hæmorrhagic Pancreatitis Type*—The attack is sudden, with violent pain in the epigastrium followed by vomiting and collapse. Tenderness and tympanites may be present in the epigastrium. Blood examination reveals subtertian parasites and quinine effects a cure.

*Pseudo-Peritonitic Type*—This is characterized by fever, pinched  
Malarial  
condition  
y chronic  
especially

when anæmia is present cancer may be suspected

### III WITH 'RESPIRATORY SYSTEM' LOCALIZATION

It will be remembered that during the ordinary attack of any malarial fever there are a few dry, rather coarse, rales to be heard when the temperature begins to fall, and earlier at the beginning of the attack there are often—as first noted by Castellani—very minute crepitations at the base probably of pleural origin which generally disappear when the temperature has reached its highest point. They both speedily disappear, but every now and then even in ordinary attacks they are more pronounced, and the patient suffers from cough or pain on taking a deep breath.

Slight as these usually are, they nevertheless are the basis of the respiratory system types of pernicious malaria, which may be classified as follows—

- 1 Pseudo bronchitic type
- 2 Pseudo-pneumonic type
- 3 Pseudo pleuritic type

*Pseudo Bronchitic Type*—This subacute or chronic dry bronchitis, with little or no fever, is cured by a few doses of quinine

*Pseudo Pneumonic Type*—This is often of the nature of a

cases broncho pneumonia in malarial patients is not due directly to the malarial parasites, but is a complication due to the pneumococcus

*Pseudo Pleuritic Type*—Intermittent pleuritic pernicious fever has been described, with sharp pricking pain, dry cough, and friction sounds, which improve in the remission and become worse again in the attack

Pleurisy of malarial origin is without effusion

#### IV WITH 'CIRCULATORY SYSTEM' LOCALIZATION

Atypical subtertian malaria with circulatory system symptoms may be classified into —

- 1 Pseudo anginal type
- 2 Endarteritis type
- 3 Intermittent claudication type
- 4 Erythromelalgia type

*Pseudo-Anginal Type*—The right heart has been noticed to be

no fever, and the blood examination may be negative, but the condition yields to quinine

At other times dry gangrene may be the only sign of an endarteritis of a deeper and more important vessel. Search should be made for malarial parasites in the blood.

in the fingers and at times the toes also. Quinine is the correct treatment

*Heart Block*—This may be due to malarial parasites

*Rarer Forms*.—Certain authors admit endocarditis as being of malarial origin

### PERNICIOUS MALARIA.

When any of the above-mentioned syndromes caused by the *subtertian parasite* become serious, and threaten to endanger the life of the patient, they are called 'pernicious malaria,' which is therefore commonly caused by *Laverania malariae*.

More rarely pernicious malaria may be due to the quartan or tertian parasites being present in enormous numbers in the blood when the syndrome usually produced is *without localization* and most frequently of the *comatose type*.

### CHRONIC MALARIA.

Chronic malaria may result from infection by any of the three malarial parasites, but is usually caused by *Laverania malariae* the subtertian parasite.

The symptoms of chronic malaria are repeated attacks of slight

symptoms are œdema about the feet, associated with anæmia which may be marked, the presence of malarial parasites in the peripheral blood during an attack of fever and often palpitation and dizziness, bronchitis and digestive troubles and a general disinclination for exertion and work. The urine shows an increase of urea and urobilin. If this state of affairs is allowed to continue it may pass into malarial cachexia with enormous enlargement of the spleen which is firm to the touch and not tender or painful, associated with a profound secondary anæmia, and great reduction of red cells and hæmoglobin, with increase of mononuclear leucocytes. In boys and girls the onset of puberty may be arrested (malarial infantilism).

In malarial cachexia the apyrexial intervals are long, during which search may in vain be made for the parasite in the peripheral blood, but it is usually found during the febrile attacks, which may be quite mild. This is a condition however, in which blood diagnosis as it does in other forms. Symptoms require to be studied, for while it may also be mistaken for

*ankylostomiasis*

Chronic malaria, of course, is due to insufficient treatment of the acute disease by quinine.

### MALARIAL RELAPSES.

feature' The reason is that the parasites are not all killed off by the quinine There are two different types of relapses the first, after a short interval may be called the true relapse, the second after a longer interval, may be called the recurrence Relapses and recurrences are probably caused by parasites belonging to the cycle of schizogony, and by the parthenogenesis of the macrogametocyte which belongs to the cycle of sporogony

Relapses of quartan fevers take place at irregular intervals, and the suggestion that they are most usual at about the twentieth day is not correct Tertian fevers relapse most commonly from the fifth to the eighteenth day, and subtertian about the end of a week after the original attack but these times are extremely variable These relapses are historically interesting as they probably represent the quintans sextans septans octans nonans etc, of ancient authors

Recurrences take place after long intervals without fever and without reinfection Exactly how long the parasites can be dormant in the human body and then wake to activity and cause fever is not known Intervals of two and three years are readily credited nowadays but it is in our experience certain that the length of time may be much longer

Parasites can certainly be found in the blood of persons long after they have left the tropics, thus, Ross mentions that they were found in a case in Liverpool four years after absence from any malarious locality, and also states that his own father suffered from attacks once or twice a year, even after nine years residence in England

It is probable that the parasites are not all killed off by the quinine

already mentioned, but especially by a chill, coming into action

### MALARIAL REINFECTIONS.

In tropical countries there are many cases of apparent recurrence

which may be apparent recurrence in these countries must be reinfections which of course may be with the same or a different species of parasite from that causing the first fever Repeated infections are the great cause of the quotidian and irregular fevers of the tropics

## COMPLICATIONS

Many other diseases may occur in the human body at the same time as an attack of malaria but of all the most important are typhoid dysentery pneumonia and nephritis

Typhoid as a complication is of course due to the *Bacillus typhosus* occurring in a person who is also infected by malarial parasites and in that sense therefore the old term typho-malaria is correct

Dysentery may be a complication due to the *Loescheria histolytica* or to the dysenteric bacilli but it may also be directly caused by the malarial parasite (Jone (p 1177)

As to whether there is or is not a malarial pneumonia is a vexed question personally we are of the opinion that a severe subtertian fever may produce symptoms resembling a pneumonia but that true lobar pneumonia when present in a malarial subject is due to the pneumococcus and is therefore a complication

Nephritis may be found in tertian and subtertian fevers being directly due to the irritation of the kidney by the malarial toxins

## SEQUELÆ

Many so called sequelæ have already been described under Atypical Subtertian Fevers (see p 1168) The possible sequelæ of malaria are very numerous and may be classified into those belonging to the nervous system and sense organs the blood the liver and the spleen

The subtertian parasite may leave severe traces of its action upon the brain after pernicious attacks and indeed the mind may never regain its old condition Apart from the milder alterations of disposition and character actual insanity in the form of mania or melancholia may result

Neuritis in some form is sometimes of malarial origin but it is quite possible that alcoholic and arsenical poisoning and indeed beri beri may have been confused with it We have seen cases of

*deafness anosmia and loss of taste* are said in some cases to be malarial in origin

have already been given in the section on Pathology to which

nuch  
liver  
but  
n a

can be met with which

ady been described, and  
s by no means unknown,

though not common, and requires prompt surgical attention. We have only come across one case in our experience

Tremors are not infrequently seen in chronic malaria, though more rare in acute malaria. *Fine tremors*, consisting of bilateral fine oscillations of small amplitude, caused by the alternate action of antagonistic muscles, may occur in the limbs and more rarely in the head. They are most common in the upper limbs, and especially in the hands. In the head there may be nystagmus or the tongue may be implicated. These tremors are increased by effort, fatigue, or emotion. *Coarse tremors* are usually exaggerations of fine tremors, and are often due to emotion, such as examination by the physician. *Intentional tremors* are produced in the hands, and sometimes in the head and neck, and are due to voluntary movement. They are not very uncommon in malarial infections.

The fine tremors must be distinguished from the fine tremors due to quinine, which are not very rare when the drug has been given for long periods. These tremors disappear in days, weeks, or months after the drug has been discontinued.

In malarial patients coming from war zones tremors and ataxic movements are not rarely seen, but these tremors are neither due to malaria nor to quinine, being of hysterical origin (*sensu lato*) and disappear often after treatment by suggestion.

### DIAGNOSIS.

The diagnosis of malaria may be simplicity itself, or, on the other hand, it may be most difficult, as there is practically *no sign or symptom of disease of the human body which it cannot mimic*. We venture to impress upon the reader the vital necessity of making a thorough and *careful clinical examination*, as in many cases this alone may be the key to a correct diagnosis.

The positive signs of malarial infections are —

1 *Tertian or quartan periodicity*, no matter what the symptoms may be

2 *Tertian, quartan, or subtertian parasites* present in the peripheral blood

be

2 *Slightly enlarge and tender spleen* in cases exhibiting syndromes usually significant of another disease, if the usual tests for the causal agent of the other disease are negative



3 *Presence of mononucleosis* in association with other features  
malaria

4 *Pigmentation of the skin*

5 *History of old malarial infections*

unless the examinations have been repeated many times and  
various intervals even after repeated  
taken quinine, a

2 *Absence of mononucleosis*

3 *Absence of enlargement of the spleen*

4 *Absence of prompt reaction to quinine*

Splenic puncture and the subsequent examination of the blood  
obtained in this way would help diagnosis considerably, but is  
devoid of risk

It is generally stated that a fe

ine therapy by various metho

L 1918 *Journal* December 7 1918

The differential diagnosis of the various forms of malarial fever  
should be confirmed no matter how evident the clinical symptoms  
may be, by microscopical examination

The most important diseases to differentiate from malaria are  
typhoid, insolation, liver abscess, kala azar, Malta fever, influenza,  
yellow fever, dengue, and seven days fever. For the differential  
diagnosis see Chapter LX (p 1511) which deals with the diagnosis  
of a tropical fever

The pernicious forms of malaria, in whatever way they attack the  
patient, will in most cases be readily diagnosed by blood examination  
as will also the masked form of the disease

Fevers due to septicæmia caused by a streptococcus, the pneumococcus,  
and the gonococcus may resemble malaria but can be excluded by bacteri-  
ological examination as can influenza when it gives rise to an intermit-  
tent type of fever

## PROGNOSIS

### PROGNOSIS

ing and they are —

- 1 The probability of recovery
- 2 The probability of a cure

1 **Probability of Recovery.**—This depends upon —

- (a) The type of fever
- (b) The condition of the patient with regard to race physical fitness the presence or absence of other diseases

dangerous

**RACE**—The mortality among natives of bad malarial usually low, while that among Europeans is high. The c in a native race which has comparatively recently been to increased danger of malarial infections is sometimes appalling

**SEX**—There is a better prognosis for males than for females there is a distinctly worse prognosis for an attack taking place during pregnancy

**AGE**—Children often have more severe attacks than adults can usually stand quinine well which rather balances the advantage

**PHYSICAL FITNESS**—Persons debilitated by long res

eg typhoid fever, etc —naturally makes the outlook more

**QUININE**—Idiosyncrasy to quinine of an anaphylactic nature is the most serious but can be combated by commencing with small doses and slowly and steadily working upwards

**DURATION OF THE SYMPTOMS**—If the symptoms persist

*Presence of mononucleosis in association with other features of malaria*

*Pigmentation of the skin*

*History of old malarial infections*

Malaria should not be excluded by

*Absence of malarial parasites from the blood* even after repeated examinations especially in people who have taken quinine and whose examinations have been repeated many times and at varying intervals

*Absence of mononucleosis*

*Absence of enlargement of the spleen*

*Absence of prompt reaction to quinine*

The question of driving the malarial parasite from a hiding place in some organ into the peripheral blood by giving a small provocative dose of quinine, injections of vaccines of sterile milk of strychnin of adrenalin by splenic chills violent exercise by ultraviolet light etc. has been attempted but success cannot be placed upon these methods as a practical aid to diagnosis. Splenic puncture and the subsequent examination of the blood obtained in this way would help diagnosis considerably but is not devoid of risk

remained unaffected while the parasite can be found in the peripheral

The differential diagnosis of the various forms of malarial fever could be confirmed no matter how evident the clinical symptoms may be by microscopical examination

The most important diseases to differentiate from malaria are typhoid insolation liver abscess kala azar Malta fever influenza yellow fever dengue and seven days fever. For the differential diagnosis see Chapter LX (p 1511) which deals with the differential diagnosis of tropical fever

by they attack the  
blood examina

micrococcus  
bacterio-  
intermittent

## PROGNOSIS.

ing, and they are —

- I The probability of recovery
- The probability of a cure

I **Probability of Recovery.**—This depends upon —

- (a) The type of fever
- (b) The condition of the patient with regard to race, age, sex, physical fitness, the presence or absence of other diseases, idiosyn-

dangerous, while pernicious malaria must be regarded as extremely dangerous

**RACE**—The mortality among natives of bad malarial regions is usually low, while that among Europeans is high. The death-rate in a native race which has comparatively recently been subjected to increased danger of malarial infections is sometimes truly appalling

**SEX**—There is a better prognosis for males than for females, and there is a distinctly worse prognosis for an attack taking place during pregnancy

**AGE**—Children often have more severe attacks than adults, but can usually stand quinine well, which rather balances this disadvantage

**PHYSICAL FITNESS**—Persons debilitated by long residence in

e.g., typhoid fever, etc.—naturally makes the outlook more serious

**QUININE**—Idiosyncrasy to quinine of an anaphylactic nature is most serious, but can be combated by commencing with very small doses, and slowly and steadily working upwards

**DURATION OF THE SYMPTOMS**—If the symptoms persist in the face of quinine therapy the prognosis is serious

2 **Probability of a Cure.**—The probability of a cure—that is to say, of a so called bacteriological sterilization of a patient with regard to the malarial parasites—is a very doubtful matter, as the

affection is able to lie dormant for months and years even after the tropics and any source of infection have been parted with entirely. At times one may be lucky enough to catch all the parasites sporulating, and to kill them with one large and properly applied dose of quinine. This has certainly happened to us with regard to the subtertian parasite in certain cases, but it may have been accidental. The infection may remain dormant for years and  
the  
In

### TREATMENT.

**Essential Treatment.**—There is one specific remedy for malaria, and this is *Quinine* which should be given *immediately* upon the diagnosis of malaria being made and may be administered as follows —

**A WHEN THE PATIENT IS FIRST SEEN IN AN ACUTE ATTACK.**

**I In Benign Intermittent Fevers** (Quartan and Simple Tertian) give *quinine in solution* by the mouth if the patient is not sick, in 10 grain doses three times a day, or in doses of 15 grains in the morning and 15 grains in the evening

If the heart is not working properly *caffeine citrate* in 1 to 3 grain doses may also be administered, either by the mouth or hypodermically

If this dosage of quinine is found to be insufficient as judged by the persistence of the symptoms *increase* it to 10 grains every four hours instead of three times a day

**II In Severe Intermittent, Remittent and All Subtertian Fevers** give *quinine* by intramuscular injection in a dose of 15 grains *as soon as the diagnosis is made*. Repeat the injection daily, and in addition administer the drug by the mouth in 10 grain doses three times a day

In these fevers as much as 45 60 grains of quinine may be required in twenty four hours to combat the symptoms and should be given by a combination of intramuscular injections with oral administration

Sodium bromide, the purgative and cardiac drugs mentioned in the preceding section may also be given

The injection is to be made in a thoroughly antiseptic manner. Use a vial containing 15 grains of quinine hydrochloride in 1 cubic centimetre of fluid manufactured by some reputable firm. The injection may be made *deep* into the muscles of the gluteal region care being taken to avoid the line of the sciatic nerve or *deep* into the loose tissue extending from the lower angle of the scapula to the crest of the ilium. The vials made by different firms vary

very much in the degree of pain or discomfort which they produce but some (a pre-war preparation) caused very little pain if given as should always be done with the strictest antiseptic precautions. For fuller instructions see Method of Administration p 1193.

Do not hesitate to give *intramuscular injections of quinine*. They have saved many lives but the strictest antiseptic methods must be used.

III *In Pernicious Malaria*—In pernicious fevers give *quinine at once by intravenous injection*.

The quinine injection should be made into the median basilic or median cephalic veins and not less than 1 gramme (15 grains) dissolved in sterile 5 c.c. of physiological salt solution should be injected at a time. The skin over the selected vein should be rendered thoroughly aseptic by

If the serious symptoms persist the intravenous injection may be repeated in four hours and further medication may then be carried on by intramuscular injections. Not more than two intravenous and two intramuscular should be given under any circumstances in twenty four hours.

When the serious symptoms abate the intramuscular injections alone should be used and should be given once or twice a day and supplemented by oral administration as indicated above.

#### B DURING THE COURSE OF THE FEVER

I *If the symptoms are abating* the dosage of quinine may be reduced slowly to 200 to 300 grains per day, 4 to 6 times a day.

II *If the symptoms are not abating* the probability is that insufficient quinine is being administered and in such cases the drug should be carefully increased.

If only 100 grains are given

In order to combat severe symptoms it may be necessary to give 45 to 60 grains of quinine or even more per diem but this must be done carefully and the dosage must at once be reduced when improvement appears. As a rule not less than 30 grains in the twenty-four hours should be given.

Quinine often takes effect before the expiration of four days of thorough treatment but it may be necessary to continue the ad-  
quinine  
must be

### C WHEN THE ACUTE ATTACK HAS SUBSIDED

Continue to administer quinine orally for at least *three months*

During the first month give it in 10 grain doses twice or thrice daily. During the second month 5 grain doses thrice daily. During the third month 5 grain doses twice daily.

The quinine may be administered as the *bis-hydrochloride* in crushed tablets but if it is desired to use the more insoluble salts (see the paragraphs on the theoretical considerations lower down in this chapter) a mixture such as the following may be given —

Quinine sulphate	10 grains
Dilute sulphuric acid	10 minims
Syrup of orange	1 drachm
Distilled water	1 ounce

Euquinine may be substituted for quinine for the purposes described in this paragraph but must be given in slightly larger doses.

With the commencement of convalescence it is useful to give some tonic mixture such as —

Iron and quinine citrate	10 grains
Liquor strychnine hydrochloratis	3-5 minims
Syrup of orange	1 drachm
Distilled water	1 ounce

This should be taken three times a day half an hour after meals. See that the patient's bowels are kept regularly open every day. Notwithstanding all care and energetic quinine treatment relapses are prone to occur some variable time after medication has ceased.

### D RELAPSES

When relapses occur after thorough quinine treatment it is advisable to combine with this drug arsenic and at times tartar emetic and phosphorus.

The following mixture introduced by Castellani indicates the line of medication suggested — Quinine sulphate 10 grains dilute sulphuric acid 10 minims tartar emetic  $\frac{1}{4}$  grain codon  $\frac{1}{4}$  grain syrup 1 drachm chloroform water 1 ounce

Another formula used by Castellani is Quin hydr gr x tartar emetic gr  $\frac{1}{4}$  liq Fowlers  $\mathbb{N}$  syr  $\mathbb{Z}$ ii ag chlorof ad  $\mathbb{Z}$ i

Two tablespoonfuls of this mixture may be taken well diluted with water every four hours.

In addition it may be necessary to resort to intramuscular injections of 15 grains of quinine daily and it is sometimes advisable to alternate this every other day by a subcutaneous injection of the *phosphorated oil* of the British Pharmacopœia in doses of from 1 to 4 minims.

The above forms the quinine phosphorus-tartar-emetiç treatment for

### E CHRONIC MALARIA

Chronic malaria should be treated by the methods given just above for relapses. Intramuscular injections of quinine are specially useful in this condition and should be given in courses of fifteen daily injections. The courses should be repeated two or three times after varying intervals.

### F MALARIA IN PREGNANCY

Give the usual twenty four hourly dosage of quinine, but divide it up into small individual doses of 2 to 5 grains.

### G MALARIA IN CHILDREN

The dosage of quinine for children is as follows —

Age of Child	Dose of Quinine	Number of Doses in Twenty four Hours
Under twelve months	$\frac{1}{2}$ to $1\frac{1}{2}$ grains	Six
1 to 3 years	1 to 2 grains	Six
3 to 10 years	2 to 3 grains	Six
10 to 16 years	3 to 5 grains	Six

### H REMARKS

It may be thought that in the above we have been too dogmatic, but our excuse is that we desire to lay before the practitioner our experience of many years of tropical practice.



FIG. 616.—TEMPERATURE CHART TO ILLUSTRATE THE BENEFIT OF INTRAMUSCULAR INJECTIONS GIVEN AT + + + + + AS COMPARED WITH THE PROLONGED ORAL THERAPY OF DAYS PREVIOUSLY.





fact that it may by this action give the liver more work by extra bile production. Consequently, it is most necessary to remind the above class of patients to take care that the bowels are open regularly, and to correct by suitable aperients any irregularity that may exist.

With regard to *mild quartan and tertian fevers*, there is not the slightest doubt that excellent effects are obtained by giving the drug four hours before the attack—i. e., before the sporulation of the parasite is due. In this way the merozoites are most likely to be killed.

one dose of 10  
dose of 20 grs

In more *severe forms of quartan and tertian*, and in cases of *subtertian fever* in which the patient is seen for the first time during the attack, the drug should be given intramuscularly or by the mouth, when it may be administered on the fall of the temperature, when the gastric irritation is lessening, and then continued by one of the above methods.

In cases of *serious and of pernicious attacks*, time must not be wasted in waiting for temperatures to fall or for symptoms to improve, otherwise the patient will die, and there must be no hesitation in giving either an intramuscular or an intravenous injection, according to the severity of the symptoms.

**METHOD OF ADMINISTRATION**—For practical purposes there are only four methods of administration—

- 1 By the mouth
- 2 By the rectum
- 3 By intramuscular injections
- 4 By intravenous injections

*By the Mouth*—If expense is no object, take the most soluble drug that can be got, and use either the bihydrochloride or the bisulphate, otherwise the sulphate must be used. In cases of women and children, equinine may be used with advantage, but it should be remembered that it is very insoluble and expensive.

The forms in which the chosen drug may be administered are—

1. Powder
- 2 Solution.
- 3 Tabloid, tablet, or pill
- 4 Cachet or capsule

It is presumed that no doctor would allow his patient to take quinine in a cigarette-paper though non medical people are found who have got into the habit of using this method

As soon as the patient is able to take

the

is

The acid preparations—for example the bihydrochloride and bisulphate—may be dissolved in water but should have some flavouring added to disguise the taste while the sulphate requires an acid which may be provided by suspending some of the powder in natural limejuice but in order to dissolve the sulphate properly of a minimum to each he unpleasant after

with apparent success in cases of chronic subtertian infection which resist the ordinary methods of administering quinine but can

for

are extremely useful for journeys being readily carried but in order to be successful they must not be old and hard The hydrochloride or bisulphate should be used and the solubility tested from time to time in water otherwise the tabloid must be reduced to powder and taken as indicated above

Pills and capsules are quite good when fresh but they are apt to get hard when old and are then useless and must be opened and the powder used if they form the only supply available

The quinine tannate chocolates are of course only used for prophylaxis in children

Quinoform suppositories (gr iii) have been used in children by Pedro and others

*Intramuscular Injections*—We are not in favour of hypodermic injections of quinine—i.e. of injections simply under the skin—

cent) saline solution, and carefully sterilized Baccelli's formula is 10 grammes of quinine and 0.075 gramme ( $\frac{1}{8}$  grain) of salt

the patient is waiting for treatment, much time is lost. We therefore recommend the tropical practitioner to purchase and keep by him the sterilettes, which are little hermetically sealed vials containing a gramme (15 grains) of quinine in solution. These sterilettes may be purchased from Squire and Sons, or Martindale, of London, Burroughs Wellcome and Co., or Molteni, of Florence. Giemsa's solution may be obtained in similar sterile vials.

The technique is simple. First cleanse the skin of the patient carefully with 1 in 40 carbolic lotion, then break off the glass seal

a little pad of wool wrung out in 1 in 40 carbolic lotion, on the site of the injection. Performed carefully in the above manner, there need be no fear of tetanus or abscess formation, and the injection

The injection should be made using a 5 c.c. or 10 c.c. syringe,

into the median basilic or median cephalic veins and not less than

dered thoroughly aseptic by soap and water and carbolic lotion or by tincture and then a bandage tied round the arm so as to retard the flow of venous blood and make the selected vein stand out. Then the needle should be inserted into the vein (care being taken that there is no air in needle or syringe) in a sloping direction with the point towards the heart so that the injection can flow with the circulation. The point of the needle should be felt to be loose (i.e. in the vein). The bandage must now be loosened and the injection made *slowly* the effect on the pulse being noted. The needle is now withdrawn and an aseptic pad fixed in position by a bandage.

**DURATION OF TREATMENT** —When the fever has subsided and the patient is feeling better the administration of quinine must not be discontinued because there is the fear of a relapse caused by parasites which have not yet been destroyed and which may be

urthenogenesis  
it may be suc

Our routine  
times a day  
three times a

day during the second month then 5 grains twice a day during the third month. In some cases when the fever has ceased it is advisable to associate *some iron and arsenic with the quinine* but these

1

may be

- 1 Symptomatic treatment of acute malaria
- 2 Diet in acute malaria
- 3 Treatment of symptoms and special conditions
- 4 Treatment of convalescence
- 5 Treatment of chronic malaria
- 6 Treatment of malarial cachexia

**SYMPTOMATIC TREATMENT OF ACUTE MALARIA** —The practitioner who works in the tropics must often be prepared to do the nursing as

k begins the patient  
well with blankets  
in case of sickness  
various stage must be  
ks hot tea etc in

order that the tox may be pu quickly as possible  
Ziemann recommends hot air baths to bring on the perspiration  
We have tried this method a few times but have not been greatly  
impressed with the advantages

The headache may be relieved by cold applications and where

there is no ice, dissolve some salt and juice of fresh limes or lemons, together with some vinegar or weak acetic acid and Florida water or eau de Cologne, in a small basin of water, and, after soaking handkerchiefs in this mixture, apply them one after the other,

patients.

When the sweating begins, the patient usually feels much better, but care must be taken to change the damp clothing, and when this stage is drawing to a close a sponge over with tepid water is most refreshing, after which he will probably go to sleep and wake

irritation albumen water

During remissions broths and clear soups and milk-puddings should be allowed. In the intermission of a quartan or tertian, and during convalescence, regular meals of good plain food may be given.

**TREATMENT OF SYMPTOMS AND SPECIAL CONDITIONS**—*Vomiting* is often distressing, and can be relieved by sips of iced soda-water or champagne. When there is no ice, cover the bottle with a thin layer of flannel soaked in water, and hang in the breeze. If these simple remedies fail, and the symptom is really urgent, apply a mustard leaf to the pit of the stomach, or give a mixture containing chloroform or a hypodermic of morphine. If, despite all this treatment, the vomiting still continues, the stomach may be washed out with slightly alkaline water.

*Hiccough* is not often present, and would be a most unpleasant

Codinae	..	gr ʒ
Syrupi toluanae		ʒi
Aquae chloroformi	..	ad ʒi.

we have used this in bad cases, we have no experience of it as a routine practice

*Diarrhœa* is at first useful in ridding the body of excess of bile and other waste products. Prolonged diarrhœa must be treated

mouth in these conditions its utility may be small

*Splenic pain and liver pain* are not as a rule severe enough to make special treatment needful but occasionally the splenic pain may be severe (due to perisplentis) when hot fomentations will

odermic saline infusions to

with hypodermic saline in the body and especially to injections consist of sterile

should be injected in one place. Oxygen inhalations if available, may also be used

*Diaphoretic pernicious fever* requires stimulants hypodermics of atropine and ether or strychnine and treatment as for algidity

*Hæmorrhagic perniosa* may be treated with calcium chloride and with local applications or injections of adrenalin

*Scarlatiform perniosa* obviously calls for dilution of the toxins by saline injections

In *pernicious cases* when the patient becomes *delirious* or coma

delirious patients not by mere causal inquiry but by percussion of the abdomen

to quinine  
joint to be

remembered in convalescence is to continue the quinine in smaller doses for at least three months after the attack. The patient

the anæmia with iron and arsenic. A course of injections of cacodylate of quinine is often useful.

**Theoretical Considerations.**—There are a number of theoretical considerations with regard to the treatment which may perhaps interest the reader and these may be divided into —

- 1 Quinine
- 2 Effects on man
- 3 Effects upon malarial parasites
- 4 Immunity of parasite
- 5 Prophylactic use
- 6 Other drugs

**QUININE**—The aim of the treatment of malaria is to kill the parasites and to aid the excretion of the toxins and relieve the symptoms of the patient. Fortunately since the days of the Countess del Chinchon the world has

fluorescence

valerianate in those who are very nervous while quinine tannate which is not nearly so bitter as the other compounds especially when made up with chocolate has been used by Celli in quinine prophylaxis for children and in cases



olved in acidulated water It has not such deleterious effects on the stomach and nervous system

Another remedy which contains quinine sulphate and which was much vaunted in days gone by is Warburg's tincture which is said to be prepared by macerating for seven days the following ingredients—

	Grams
Aloes socotrinae	240
Rhei	80
Fructi archangelicæ officinalis	80
Radici inulæ beleni	40
Croci	40
Fructi foeniculi	40
Cretæ preparatæ	40
Radici gentianæ	20
Radici curcumæ zedoariæ	20
Cubebæ	20
Myrrhæ	20
Polyponi officinalis	20
Opi	2½
Piperis nigræ	4
Cinnamoni	3
Zingiberis	8

in a pint of 60 per cent alcohol for a week then pressing filtering and dissolving in the filtrate —

	Grams
Quininae sulphatis	175
Camphoris	20

and after three days filtering making up to the pint with alcohol The dose

depressant producing a fall in the arterial pressure with decrease in the

cardial eruptions

*Quinine Hemoglobinuria*—In persons who have suffered from malaria

processes in permanent cases (see also p. 2007)

*Quinine Fever*—In latent malaria a small dose of quinine may occasionally provoke a febrile paroxysm. This has been compared to the action of a small dose of salvarsan in provoking an exacerbation of symptoms in

EFFECT UPON MALARIAL PARASITES—Quinine appears to particularly

be present in the blood in a strength of at least 1 in 20,000. According to Thomson, though quinine has no direct destructive action upon the crescents

relapse during thorough quinine treatment. In these cases the quinine dosage must be increased. We have administered 2 grammes by intramuscular

doses of quinine have often to be increased in order to cure an attack of fever in the individuals who have taken quinine more or less irregularly and that persons who have taken quinine regularly when in an endemic area may have an attack of malarial fever after leaving this area and ceasing the drug.

**PROPHYLACTIC USE**—This will be discussed in the section on Prophylaxis.

**OTHER DRUGS**—It is almost a work of supererogation to mention other drugs in the treatment of malaria such as cupreus mercury atoxyl and treatment by the serum of immune animals and by violet light or in the dark. Methylene blue however has been used by several authors in the dose of 2 grains every four hours. In our experience its efficacy cannot be compared to that of quinine.

Surveyor has recommended the administration of 2 grains of picric acid twice or three times a day by the mouth as a method of destruction for the crescents of *L. malariae*. The drug can also be administered by injections of sodium picrate. Nicolle and Conseil and more recently Falconer, Anderson, Micheli, Quarelli and others have tried salvarsan in malaria with only moderate results. If used it must be combined with quinine. Neosalvarsan has been found to be useless in subtertian fevers but it and salvarsan may act upon *Plasmodium vivax* intravenously or intramuscularly. For dosage see p. 1313.

Tartar emetic has been used by Rogers but the researches by Low and others have shown that when administered alone it has no effect upon the malarial parasites. Röntgen therapy is useless though the spleen may get smaller.

## PROPHYLAXIS

The very great success which has followed every serious attempt at prophylaxis undertaken during the last few years has made it the urgent duty of each community to scientifically apply a well thought out scheme of a not too expensive nature to its district

that malaria depends upon—

- 1 The presence of numerous human beings infected with male and female gametocytes
- 2 The presence of numerous anophelines in which the gametocytes are capable of developing into sporozoites
- 3 Free access of these anophelines to the infected human beings

- 4 Air-temperatures suitable for the development of the malarial parasite in man and anophelines
- 5 Free access of infected anophelines to non immune human beings

To summarize there are three factors (1) Man, (2) the anopheline, (3) the air temperature

In the tropics this third factor is in favour of malaria, and it cannot be altered therefore methods of prophylaxis must be devoted to the human being and the anopheline

Man—The preliminary step with regard to man is to ask his intelligent assistance, and for this purpose education with regard

First  
touch  
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cated public of the tropics of the monetary loss caused to the Government, the planter, the merchant—to the employer of labour—by malaria Unless this can be done the money necessary for the effective prophylaxis will not be forthcoming No one will deny that malaria is the greatest cause of sickness in the tropics though there may be places where its mortality is low and places where it is high, but that which people fail to recognize is the financial loss caused by this sickness

Therefore we quote the well known example of the Adriatic Railway Company, which, according to Ricci used to spend on account of malaria among 6,416 workmen living in malarious areas

pamphlets in the vernacular, written in simple language, reviewing the subject of the lecture, together with, if possible, the demonstra-

In such instruction no pamphlet or book should be used, otherwise there is a danger of the knowledge being acquired by rote. The only way, however, to bring about any result by this method is to award a special grant to the school, based upon the knowledge of the scholars.

All these methods have actually been and are being carried out, but in any case they are only preliminaries, and any good which may result therefrom will not be immediate.

Another preliminary matter is to use the knowledge acquired concerning the life-history of the anophelines and not to build

Negri, and others

Koch's method consists in taking a large dose (15 grains) of quinine on two consecutive days every eight or ten days for three months, while Celli's method consists in taking about 6 grains (0.40 gramme) of quinine daily, Plehn's, in taking 8 grains of quinine every fourth day. Our own method is to give 5 grains daily, and,

by Watson, or chocolates of tannate of quinine (2 to 5 grains), as advised by Celli, may be administered.

In very malarious zones such as certain tropical countries and the Balkans, the doses mentioned are not sufficient and should be increased.

but... rise  
after... our  
experience is a tremor of the hands, but various other nervous symptoms may appear—*e.g.*, irritability

The advantages of quinine prophylaxis are, however, in excess of its disadvantages, and we strongly recommend its use. In Italy special laws have been passed, due to the efforts of Celli, which render quinine prophylaxis practicable among the poorer classes. The quinine is manufactured by the State and distributed gratis to the poor, while employers of labour are compelled to supply it to their work-people. Governments, municipalities, etc., can also help in this way by giving free quinine to the populace.

The systematic free distribution of quinine powders, tinted if

cate also presence of various other substances

By this method Koch freed Stephensort, in New Guinea from malaria, but there is a doubt as to whether the disease will not recur if this method alone is used. It must be remembered that free quinine distribution on a large scale is very costly.

*Bite Prevention* —

The next method is to prevent man from being bitten by anophelines. This may be



FIG. 627.—THE SIMPSONETTE DESIGNED TO PROTECT THE WEARER FROM MOSQUITO BITES (From a photograph given us by Mrs Mary Simpson.)

It is also advised not to go out after sunset, and to protect the whole body against bites.

Firstly, with regard to the mosquito-curtain the mesh must be suitable—*e.*, twenty to twenty four meshes to the square inch. The lower part of the net should be tightly tucked in under the mattress or loosely or

mulate all sorts of malarious region

without your own net, as the condition of those in rest houses may

or houses, these can using wire-netting of twenty to twenty four meshes to the inch, preferably the latter

supporting the gauze, and have a double door. They are only 12 feet by 12 feet by 8 feet, and can therefore be erected inside the room of a native house if desired, or on a veranda. The cost is small.

Hospital wards ought certainly to be rendered gnat-proof in malarious districts as persons are often found in the wards with their blood teeming with gametocytes. Hence if there are any suitable anophelines in the hospital the chance of future patients suffering from other diseases becoming infected is considerable.

With regard to coolies the third method has been tried to a certain extent with success—viz, that of rubbing all over the body an oil composed of  $1\frac{1}{2}$  parts of citronella oil, 1 part of kerosene, and 2 parts of coconut oil, to which 1 per cent of carbolic acid is added. Coolies seem to like this mixture, which is called the 'bamber green oil'. Menthol, cinnamon oil, eucalyptol, camphor ointments or powders may be used.

Electric fans and punkahs are secondary methods of preventing anophelines from biting human beings.

**SULPHUR**—According to some authorities mosquitoes and flies will not bite persons who take small doses of sulphur regularly but this is not so in our experience.

The healthy may also be segregated as far from sources of infection as possible. This may be temporary or permanent, according to local conditions.

**The Anophelines**—It has already been indicated that it is not necessary to exterminate the anophelines in order to prevent malaria, but that all that is needful is to reduce their numbers. The objection has been raised to anopheline destruction that as fast as they are destroyed in a given area others will immigrate into that place from surrounding areas.

Ross has rather aptly answered that objection by pointing out

of the density surrounding that area. A square one mile wide will have a central density of 2 per cent., at a quarter of a mile from the boundary 11 per cent., and at the boundary 50 per cent. of the surrounding density. No experiments have, however, been carried out to determine whether these assumptions are correct.

There is, however, no doubt that anopheline reduction leads to the reduction of malaria. The work done under Ross's direction

admitted into the hospitals from 610 in 1901 to 23 in 1905, while the cases from the surrounding district in which no antimalarial methods were employed were 197 in 1901 and 353 in 1905. The children found infected in the two towns in 1905 amounted to 0.5 per cent. of those examined, while in the surrounding district they were more than 23 per cent. The financial saving is also shown by the fact that Government employes in 1901 obtained 236 sick certificates, amounting to 1,026 days' leave, and in 1905 only 4, amounting to 30 days' leave. The cost to the end of 1905 was £10,100, with a yearly expenditure of £410. The cost up to the end of 1905 worked out at £1.48 per head of the population.

Simplex (1907) has been determined by Ross (1901) that the destruction of anophelines will lead to the diminution of malaria. There can therefore be no doubt that the destruction of anophelines will lead to the diminution of malaria.

*The Insect*—The measures which are used may be classified into those directed against the insect and those against the larvæ. With regard to the insects, fumigation with some substance such as sulphur (1907) has been determined by Ross (1901) that the destruction of anophelines will lead to the diminution of malaria.

by Ross and Gorgas, while the use of a hand-fan is also recommended. Mosquito traps have also been used and Giemsa's



A. *Engineering works intended to eliminate breeding places:—*

- 1 Draining swamps
  - 2 Draining roads
  - 3 Filling in hollows
  - 4 Training streams
  5. " " " " " "
  6. " " " " " " this is  
down
- of dense plantations until the trees are at intervals, and the removal of trees with rot holes, which, however, may be filled
- 7 Segregation either of the whole community, by altering the residences from some very infected area or partial segregation of a chosen portion of the community from the worse-infected portion Simpson recommends that at least a zone of 300 yards in width, preferably 400 yards should separate the residential European quarters from the Native town Since segregation has been introduced in West Africa, a considerable improvement in the health of the European community has taken place
  - 8 Efficient drainage to keep down subsoil water-level.

B. *Sanitary works —*

- 1 *Removal of small collections of water* Regular inspection of compounds and careful collection and disposal of household utensils likely to harbour mosquito larvæ Repair of house gutters
  - 2 *Oiling collections of water with kerosene (crude petroleum) by means of sprays* every ten days, 1 gallon being allowed for 1,000 square yards
  - 3 *Larvicides* — Measure the length and breadth of the area of water and add 1 in 20,000 to 1 in 50,000 sanitas okol or 1 in 40,000 120-12al, or the Panama disinfectant
4. *Screening wells, cisterns, cesspits, etc., with wire gauze*
- 5 *Removal of plants likely to contain water and act as*

It is always easy to find some local fish which will eat larvæ readily. Thus, in Bengal the larvæ-eating fish are *Haplochilus panchax*, *H. melastigma*, *Ambassis nama*, *A. vanga*, *Anabas scandens*, *Barbusicto* and several species of *Trichogaster*. Tadpoles and water-boatmen also eat mosquito larvæ.

7 *Growth of weed* in water, especially *Lemna*, the duckweed.

8 *Attention* to the non stagnation of water in gutters

**Summary.**—Every district must be considered separately, and a systematic malarial survey having been made, those methods should be applied which seem most adapted for success. A combination of methods must be better than any one individual method. In any case a definite scheme should be devised, and thoroughly and continuously carried out. But a word of caution is necessary. Having made the survey and estimated the cost, firstly, leave a good margin for contingencies, and, secondly, insist upon an adequate supply of money being provided, but it is not necessary to embark upon an expensive scheme, as a great improvement can be effected with but little expenditure. It is, however, important

cides, by natural enemies, or inconvenienced by diminution of dense vegetation

Larvæ may be attacked by removing, oiling, or screening col-

RELATIVE VALUES OF QUININE PROPHYLAXIS AND ANTIMOSQUITO PROTECTION

Method of Prophylaxis	None	Quinine Prophylaxis alone	Anti mosquito Protection alone	Quinine Prophylaxis plus Antimosquito Protection
Percentage of infections ..	33%	20%	2.5%	1.75%

**Results.**—In British Guiana the cases treated in hospitals have been reduced from 33,748 in 1906-07 to 7,384 in 1912-13. In the

Panama Canal Zone the death rate from malaria in 1881 was 20.5 per 1 000 in 1911 0.96 per 1 000. The malaria cases in 1906 were 821 per 1 000 and in 1911 184 per 1 000. In Ismailia in 1900 there were 2 284 cases in 1906 0.8 not a case. In Kuala Lumpur the death rate was 9.7 per 1 000 in 1907 and 3.9 per 1 000 in 1914. These figures speak for themselves.

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## CHAPTER XXI

# THE TROPICAL HÆMOGLOBINURIAS

General remarks—Malarial hæmoglobinuria—Quinine hæmoglobinuria—  
Blackwater fever—References

### GENERAL REMARKS

HÆMOGLOBINURIA or the presence of more or less altered hæmo-  
globin in the urine is found in a number of conditions which may

high occasionally occur  
Raynaud's disease the

2 The Toxic Hæmoglobinurias, which are brought about by the  
action of a number of drugs as for example quinine and its salts  
chlorate of potash antipyrin carbolic acid and naphthol and by  
certain vegetables used as food as for example certain beans  
(Favism *vide* p 202)

3 The Specific Hæmoglobinurias, which are specific diseases such  
as blackwater fever and paroxysmal hæmoglobinuria

In the tropics we are concerned with one entity in each of these  
groups—viz —

- 1 Malarial hæmoglobinuria
- 2 Quinine hæmoglobinuria
- 3 Specific blackwater fever

### 1 MALARIAL HÆMOGLOBINURIA

Definition Malarial hæmoglobinuria is a condition in which hæmo-

globin is present in the urine occurring in the course of atypical sub-  
tertian malaria and caused by the malarial parasites Stephens  
has seen it associated with quartan parasites

**Ætiology**—The ætiological factor is *L. malariae* Grassi and Fel-  
letti 1890 together with some other factor which inhibits the  
production of antihæmolysins

**Climatology**—The distribution is coextensive with the distribu-  
tion of the more severe forms of malaria and is therefore most  
evident in the tropics and subtropics

**Pathology.**—As has been shown by de Blas, Brem, and Zeiler, the malarial parasites give rise to a hæmolysin which probably varies in quantity and quality with different strains of parasites, but is kept in check by the action of anti-hæmolysin, which is formed in the body, but which under certain circumstances—e.g., exposure to the weather, etc.—may fail to be produced in sufficient quantities and hæmoglobinæmia with hæmoglobinuria may occur. Brem found that three parts of a hæmolysin extract from a case of pernicious malaria completely destroyed (hæmolyzed) one part of a 5 per cent suspension of erythrocytes in twenty minutes. This hæmolysin is thermolabile. Zeiler and Brem have also demon-

Bijon considers that the resistance of the red corpuscles to lysis is diminished, and Gasbarrin believes that the lysin lies inside the red cells.

## 2. QUININE HÆMOGLOBINURIA.

tion of a 9 grain dose of quinine was followed by hæmoglobinuria in a few hours. Ketchen has recorded a case of seven consecutive

hæmoglobinurias in the same individual in whom each attack was the sequel to a dose of quinine. We have met with similar cases, but our maximum is six attacks in one year

**History.**—Veratas in Greece, in 1858, was the first to draw attention to this form of fever, and to definitely ascribe the hæmoglobinuria to quinine, and he was followed and supported by Tomaselli Grocco and many other Italian and Grecian observers. Later Plehn, Koch, and others have strongly advocated this theory, but these last observers have applied this one hypothesis for the explanation of all the conditions included under the term 'blackwater fever'

**Climatology.**—The attack can take place anywhere for the first time provided that the individual is suffering from chronic malaria, etc., and the unknown factor or factors to be mentioned below, and has taken the requisite dose of quinine

**Ætiology.**—The causation of this condition is the administration of quinine in cases of malarial cachexia and chronic malaria, but this is not the entire ætiology, otherwise the condition would be more commonly met with than at present, and also it is quite safe to administer quinine to the majority of cases of chronic malaria and malarial cachexia without causing hæmoglobinuria. Moreover, the administration of a dose of a salt of calcium prior to the quinine will prevent the hæmoglobinuria which in the same individual has occurred after such administration

From one observation which we have made we would throw out the suggestion that one of the other factors in quinine hæmoglobinuria may be the condition of the kidney and that the site of the hæmolytic may be in that organ

**Pathology.**—The pathology of quinine hæmoglobinuria is but little understood, but it has been very ably studied by Barratt and

the same

3. The hæmolytic produced by quinine (alkaloid) resembled a

the drug

With regard to the action of the quinine, some observers believe that it produces the hæmolytic by lowering the osmotic pressure of



**Pathology.**—As has been shown by de Blasi, Brem, and Zeiler, the malarial parasites give rise to a hæmolysin which probably varies in quantity and quality with different strains of parasites, but is kept in check by the action of antihæmolysin, which is formed in the body, but which under certain circumstances—*e.g.*, exposure to the weather, etc.—may fail to be produced in sufficient quantities and hæmoglobinæmia with hæmoglobinuria may occur. Brem found that three parts of a hæmolysin extract from a case of pernicious malaria completely destroyed (hæmolyzed) one part of a 5 per cent suspension of erythrocytes in twenty minutes. This hæmolysin is thermolabile. Zeiler and Brem have also demonstrated the presence of antihæmolysin in the serum of normal individuals as well as in that of persons suffering from pernicious malaria. It would therefore appear as though the presence or absence of hæmoglobin in an attack of pernicious malaria depends upon the relationship between the quantity of hæmolysin produced, and the amount of antihæmolysin also produced.

Bijon considers that the resistance of the red corpuscles to lysis is diminished, and Gasbarrini believes that the lysin lies inside the red cells.

**Symptomatology.**—The symptoms are those of an attack of pernicious malaria, in which the main feature is the presence of hæmoglobinuria, the other symptoms being high fever, shivering, vomit-

“  
 .  
 demonstrating  
 the rarity of

**Treatment**—This is the same as for other forms of atypical sub-

!"

## 2. QUININE HÆMOGLOBINURIA.

**Definition.**—An acute non contagious fever caused by the administration of any of the ordinary salts of quinine in certain cases of malarial cachexia and chronic malaria, and characterized by hæmoglobinæmia and hæmoglobinuria.

**Remarks**—Sir Patrick Manson has pointed out that an attack of hæmoglobinuria can be produced in certain cases by the ingestion of a single small dose of quinine. Ross and Low have reported such a case under the care of Sir Patrick Manson where the administration of a 9 grain dose of quinine was followed by hæmoglobinuria in a few hours. Ketchen has recorded a case of seven consecutive

hæmoglobinurias in the same individual in whom each attack was the sequel to a dose of quinine. We have met with similar cases

Plehn Koch and others have strongly advocated this theory but these last observers have applied this one hypothesis for the explanation of all the conditions included under the term blackwater fever

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**Pathology**—The pathology of quinine hæmoglobinuria is but

THE SAME.

3 The hæmolysis produced by quinine (alkaloid) resembled a

THE SAME.

With regard to the action of the quinine some observers believe that it produces the hæmolysis by lowering the osmotic pressure of the blood plasma.

**Morbid Anatomy**—We are not acquainted with any direct observations on this subject but the appearance of the kidney in people who have died from pernicious malarial fever in which large doses of quinine have been administered without success resembles

**Pathology.**—As has been shown by de Blasi, Brem, and Zeiler, the malarial parasites give rise to a hæmolysin which probably varies in quantity and quality with different strains of parasites, but is kept in check by the action of anti-hæmolysin, which is formed in the body, but which under certain circumstances—*e g*, exposure to the weather, etc.—may fail to be produced in sufficient quantities, and hæmoglobinæmia with hæmoglobinuria may occur. Brem found that three parts of a hæmolysin extract from a case of pernicious malaria completely destroyed (hæmolyzed) one part of a 5 per cent suspension of erythrocytes in twenty minutes. This hæmolysin is thermolabile. Zeiler and Brem have also demonstrated the presence of anti-hæmolysin in the serum of normal individuals as well as in that of persons suffering from pernicious malaria. It would therefore appear as though the presence or

red cells

the presence of *L. malaris* in the blood, and then by the rarity of severe jaundice

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**Remarks.**—Sir Patrick Manson has pointed out that an attack of hæmoglobinuria has been reported such as the administration of a 9 grain dose of quinine was followed by hæmoglobinuria in a few hours. Ketchen has recorded a case of seven consecutive

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**History.**—Veratas in Greece, in 1858, was the first to draw attention to this form of fever, and to definitely ascribe the hæmoglobinuria to quinine, and he was followed and supported by Tomaselli Grocco, and many other Italian and Grecian observers. Later Plehn, Koch, and others, have strongly advocated this theory, but these last observers have applied this one hypothesis for the explanation of all the conditions included under the term 'blackwater fever.'

**Climatology.**—The attack can take place anywhere for the first time provided that the individual is suffering from chronic malaria, etc., and the unknown factor or factors to be mentioned below, and has taken the requisite dose of quinine.

**Ætiology.**—The causation of this condition is the administration of quinine in cases of malarial cachexia and chronic malaria, but this is not the entire ætiology, otherwise the condition would be more commonly met with than at present, and also it is quite safe to administer quinine to the majority of cases of chronic malaria and malarial cachexia without causing hæmoglobinuria. Moreover, the administration of a dose of a salt of calcium prior to the quinine will prevent the hæmoglobinuria which in the same individual has occurred after such administration.

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3. The hæmolysis produced by quinine (αλκαλοϊδ) resembled a

the drug

With regard to the action of the quinine some observers believe that it produces the hæmolysis by lowering the osmotic pressure of the blood plasma.

**Morbid Anatomy.**—We are not acquainted with any direct

both macroscopically and to some extent microscopically the kidney seen in blackwater fever

**Symptomatology.**—The general symptoms resemble those of an attack of blackwater fever, but are not so severe, and the jaundice is slight or absent

**Diagnosis.**—The history of the attack following the administration of quinine in persons suffering from malaria cachexia or chronic malaria may give a clue

of the time

**Prophylaxis.**—Europeans about to visit or reside in the tropics should be given a test dose of 10 or 15 grains of quinine by the mouth, in order to exclude idiosyncrasy. In cases of chronic malaria or malarial cachexia, in which this condition may appear, calcium lactate in 10 gram doses should be given before each dose of quinine

## B SPECIFIC BLACKWATER FEVER.

*German* Gallenfieber Schwarzwasserfieber

**Definition.**—Blackwater fever *sensu stricto* is an acute specific fever of unknown causation, characterized by the severity of the symptoms, great blood destruction, jaundice, and hæmoglobinuria

**History.**—The knowledge of this fever is recent, for it does not appear to have been noted by Torti, the celebrated writer on pernicious fevers, nor by his predecessors, and the first information of its existence appears to have been given by the French naval surgeons Lebeau, Daullé, and Le Roy de Mericourt in 1850-53, who drew attention to the disease in Madagascar and Nosse Be, after which it is mentioned as occurring in Senegal, Cayenne, and

the Antilles. In 1858 Veratas described its occurrence in Greece,

by Barthélemy and Benoît in 1865; Bérenger-Féraud in 1874 (in which year Tomaselli first described it in Italy), Pellarin in 1876, and O'Neill in 1882. Kelsch and Kiener in 1889 gave an

seen on the Gold Coast, where it is called *attridi assara*, which means 'bilious fever.' The name 'blackwater fever,' now universally adopted, was, as far as we know, first used by Easmon.

Researches have been made as to its nature and treatment by Koch, Plehn, Crosse, Prout, Stephens, Christophers, Bentley, Barratt, Yorke Cardamatis, Leishman, Low and Wenyon, Balfour, and others.

1. *Common type* — Very slight icterus, anuria, coma, death in twenty-four hours.

2. *Common type* — Very slight icterus, anuria, coma, death in twenty-four hours.

3. *Fulminating type* — Very slight icterus, anuria, coma, death in twenty-four hours.

4. *Common type* — Very slight icterus, anuria, coma, death in twenty-four hours.

5. *Common type* — Very slight icterus, anuria, coma, death in twenty-four hours.

quinine

It is found in South India, Ceylon, Madras, and

Terai, Assam, the Jeypore district of Madras, and the Canara district of Bombay. It is also found in China, Cochin, China, and Farther India.

of the U

Brazil—

Sudan, but there are many other parts of the tropics from which it has not been reported. Too much trust must not be placed upon this distribution, as confusion exists between the mild attacks of quinine hæmoglobinuria, as well as the atypical subtertian malarial form. True blackwater fever can exist in epidemic form and is a very fatal infection.

**Ætiology**—The causation of blackwater fever has been much complicated by the confusion arising from the non-recognition of quinine and malarial hæmoglobinurias, but when these conditions are admitted there is still the serious disease, blackwater fever, to be explained, and its causation appears to us to be some protozoal parasite as yet unknown.

The various theories which have been advanced to explain the ætiology of blackwater fever are—

- 1 The malarial fever
- 2 The theory of malaria together with some other factor
- 3 Malarial anaphylaxis theory
- 4 An unknown agent theory
- 5 Bite of an unknown arthropod

1 *The Malarial Theory*—All the old writers on the ætiology of black

tropics

93.8 per cent. Further the presence of a hæmolyxin in malaria has been

attacks insufficiently combated by quinine. In such cases of chronic malaria  
in those suffering from anæmia with repeated attacks of fever and  
repeated doses of quinine—blackwater fever sooner or later almost certainly  
supervenes—at least in tropical climates.

These statements are too sweeping if genuine blackwater fever is meant  
otherwise the home of the disease would be Ceylon whereas it is so rare that  
we have never heard of a genuine non imported case for in this island there  
are Europeans and natives with just the conditions required by Stephens  
and yet they do not develop blackwater fever because the only two cases  
which we have met with or heard of in Ceylon in twelve years were most prob-

and the less frequent occurrence in other parts of India where malaria is  
common



produce the requisite conditions they maintain that there must be a population non immune to malaria of which infection there must be a large portion due to *L. malaris* and malaria must be in such quantity as to produce an almost continuous infection and this must be associated with a neglect of

4 *Unknown Agent*—Sir Patrick Manson, in 1893, first promulgated the theory that blackwater fever was a disease distinct from malaria, and supported this by the peculiar distribution of the disease in tropical Africa and very local in India.

In our opinion blackwater fever is a disease of its own though

its distinction is therefore probably due to some acquired or inherited immunity. It is said that a person is usually not attacked until

and other disease—e.g. malaria symptoms—or certain drugs.

Pathology—As the etiology of the disease is uncertain the pathogenesis is also little understood.

*Christophers and Bentley* have brought forward an explanation of the pathology. The view is that

They, supported by de Haan, consider that the suppression of

which tends to lower the blood pressure, and by that means the secretion of water by the glomeruli, but that if the blood-pressure is kept up by the injection of saline solutions, the tendency to suppression is decreased. This is of importance in guiding the treatment of the condition.

**Morbid Anatomy.**—There are three cardinal features in the

hæmoglobinæmia and chokæmia, with lessened tonicity. The red cells and hæmoglobin are greatly reduced, and the former may include shadow cells and small fragments of cells, and deep staining round cells (spherocytes) in the early stages, and later may show

cytosis with polymorphonuclear and mononuclear increase but when the fever disappears there is leucopenia with a mononuclear increase

seen. Bile pigments are seldom present and may be recognized by Gmelin's or Marechal's reactions. There is a considerable amount of albumen present in the form of serum albumen, serum globulin and nucleo albumen. Phosphates are said to be diminished. The hamozotic value is higher than that of the red corpuscles of the blood.

usually the patient complains of lassitude, pains all over the body,

legs and an intense feeling of weakness and nausea which as a rule quickly ends in retching and then vomiting first of food and then of green bile. The tongue is coated with a dirty yellowish fur and there is much thirst and constipation the feces at first being

**General Treatment**—In treating the disease the important features to be remembered are that the patients are often infected

*anuria and uræmia* Lastly the disease is apt to relapse

From the very commencement the patient must be put to rest  
 15 to  
 water  
 by the

mouth use soda water albumen water whey cold or warm tea barley water or toast water in quantity If vomiting is such that liquids cannot be retained by the stomach use rectal enemata of warm physiological saline solution (0.9 per cent of common salt in water) or *sterile* subcutaneous injections (temperature 98.4° F or 37° C) of a mixture such as the following —

Calcium chloride	4.5 grammes
Sodium chloride	10 grammes
Distilled water	1 000 c c

One hundred to two hundred cubic centimetres of this mixture, *properly sterilised* may be used two or three times a day as a subcutaneous injection in bad cases

**Diet**—The diet must be fluid preferably in the form of whey

indicate stimulants at times but there is no doubt of the value of champagne and brandy when they can be administered

**Symptomatic Treatment**—*Vomiting* may be relieved by sips of iced or cooled soda water or champagne If these simple remedies fail apply a mustard leaf to the pit of the stomach Tincture of iodine in a strength of 1 to 2 drops in an ounce of cinnamon water and administered orally several times a day may be useful If this fails hypodermics of morphine must be tried but it must be admitted that we do not like to administer this drug in blackwater fever unless compelled and prefer to combine atropine with it and even then to give as little as possible *Constipation* may be combated by means of calomel in repeated small doses helped if necessary by enemata *Diarrhœa* should not be too rapidly stopped but if it is

a pressing symptom, weakening and disturbing the patient, then tannic acid in 15 grain doses, tannalbin in 15 grain doses, or bismuth subnitrate in 10-20 grain doses, may be administered by the mouth, or enemata of tannic acid given by the rectum if there is also much vomiting.

The *heart's action* should be carefully watched, and may require support by hypodermic injections of digitalin or caffeine. Calcium lactate, having a tonic effect upon the heart, and being also useful for other reasons in this disease, may be given in 10 grain doses twice or three times a day, or in the form of the injection mentioned above. Extract of the pituitary gland has been recommended in cases of cardiac failure.

*Malarial parasites*, when present in the blood require treatment by intramuscular injections of *quinine hydrochloride* (p. 1188), preceded by a dose or two of calcium lactate, but the sulphate or bisulphate of quinine are contraindicated, and should not be employed.

*Pain in the back* should be treated by hot fomentations and if these fail and the symptoms be urgent, by morphine given subcutaneously.

*Anuria* must be met by vapour baths, before which a hypodermic injection of *pilocarpine* may be given. If this fails, dry or wet

special drugs must not be given.

2. **AFTER THE ATTACK**—If the patient survive the attack of true blackwater fever, which is often fatal he should be allowed a long convalescence, with a change to a temperate climate if possible. During this time he will require good, wholesome, nourishing food and slowly graduated exercise.

One attack of 'blackwater fever' *per se* should not necessarily

dangerous

3. **REMARKS**—Such is the outline of the treatment which we advise, but other authors have different views, and one or two of these may be briefly mentioned—

**Prophylaxis**—Very little can be said with regard to the prophylaxis as the knowledge of the aetiology is incomplete. As black water fever generally develops in persons who have suffered from malaria, quinine prophylaxis should be carried out in the manner already described in the chapter on Malaria (Chapter XL p 1204) attacks of malaria being treated by quinine tannate in small repeated doses or the drug should be preceded by a dose of calcium lactate.

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## CHAPTER XLII

# YELLOW FEVER

Synonyms—History—Etiology—Pathology—Symptomatology  
—Diagnosis—Prognosis—Treatment—Prophylaxis—References

**Synonyms**—Bilious Remittent fever Acclimatizing fever, Inflammatory fever Febris Flava Pestis Americana Typhus Icterosus *French* Fièvre Jaune *Italian* Febbre Gialla Febbre Amarilla *German* Gelbfieber In

of leucocytes and polymorphonuclear leucocytes It is spread by

P. du Tertre, in Guadeloupe, in 1635, but it soon became well known from the epidemic in Cuba in 1648-49, Jamaica, 1655, San Domingo, 1656, Martinique, 1688 and Vera Cruz 1690. In 1698 it was recognized that the disease was being carried from place to place by ships, and a quarantine ordinance was brought into force, which lasted from 1709 till 1790, when the extensive wars of the period caused it to be ineffective with the result that between 1791 and 1815 most extensive epidemics took place, and gave



towns during yellow fever epidemics though rare in preceding years With regard to bilious remittent fever a good account of the volume of the *Medical Journal* t or mosquito of being the possible carrier of yellow fever In 1876 Dowell of Galveston showed that mosquitoes and yellow fever obeyed the same natural laws and in 1878 it was demonstrated in Mobile that quarantine of the patients together with sulphur fumigation could control the epidemic But it was not till 1881 that Charles Finlay of Havana directly attributed the spread of the disease to the mosquito In 1882 Gerard having caused a mosquito to suck the blood of a patient on the fourth day of the fever then allowed it at once to bite his hand with the result that he developed in due course a mild attack of yellow fever The credit of having years in numerous publica

ight that he had discovered of a micrococcus and later C Valle C Finlay and Gibier each described specific bacteria Sternberg studied the disease for years but could find no definite bacterial or other cause He however in a certain number of cases came across a bacillus which he called  $\lambda$  In 1897 Sanarelli announced that he had found a bacterium (*Bacillus icteroides*) which he believed to be the cause of the disease and further he prepared a serum for its treatment Sanarelli's findings were at first confirmed by a large number of observers

species although it plays no part in the ætiology of the malady and is merely the cause of a secondary infection In the same year Reed Carroll Agramonte and Lazear proved that the disease could be produced by the subcutaneous injection of infected blood into a non immune person that the disease was not contagious

In 1903 Parker Beyer and Pothier concluded that the ætiological cause was *Myxococcidium stegomyia* found in infected mosquitoes which they

In 1905 Rosenau Francis and Beyer showed that the disease could be communicated by the inoculation of infected blood filtered through the closest grained Pasteur Chamberland B filter which

they could obtain and therefore came to the conclusion that the causal agent of the disease must be of ultramicroscopic size

In 1909 Seidelin described the presence of minute bodies (*Paraplasma flavigenum*) in the red cells of persons suffering from yellow fever but these are not believed to have anything to do with yellow fever. Low and Wenyon have shown that Seidelin's bodies are common in the blood of young guinea pigs

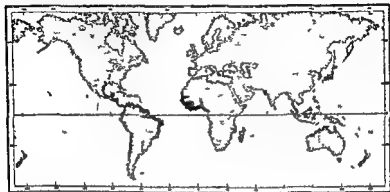


FIG 628 —MAP OF THE DISTRIBUTION OF YELLOW FEVER (After Newstead)

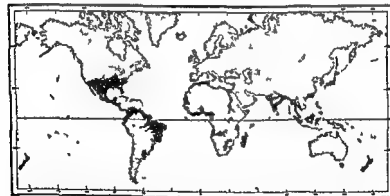


FIG 629 —THE DISTRIBUTION OF *Stegomyia calopus* (After Newstead)

The theory of the propagation of the disease by *Stegomyia calopus* has been put to the practical test of prophylaxis with most

Mexico and New Orleans and  
The late Sir Robert Boyce  
nition to the yellow fever of  
leddy at times, and which is

mosquito.

**Climatology.**—As already stated, the endemic area includes a portion of the Atlantic coasts of Mexico and Central and South America, as well as the Antilles. Some of the more important endemic centres at the present time are Guatemala, Spanish Honduras, Nicaragua, Costa Rica, Salvador, French Guiana, Dutch Guiana, along the Rivers Magdalena, Orinoco, and Amazon, and in Ecuador. There is evidence that the Cape Verde Islands were infected in 1510-15, and that the Gulf of Benin received its yellow

fever, but, though these early historical points cannot satisfactorily be settled, there is more evidence that the true home is Central America, and that Africa is a secondary endemic area, in which very serious outbreaks have been recorded in the past, and where the disease is at times epidemic.

From the endemic centre the disease can spread by the agency of ships until it reaches  $46^{\circ} 56' N$  (Quebec) and  $34^{\circ} 54' S$  (Monte Video), which correspond to the isotherm of  $60^{\circ} F$  ( $16^{\circ} C$ ) for the mean temperature of the hottest month of the year. At its northern limits the disease is generally very mild, but at its southern limits it may be very severe.

It appears to us that the fever associated with black vomit found among children in 'Grande terre' Guadeloupe must either be a form of yellow fever or closely allied to it though possibly some cases may be confused with the

Yellow fever has been recorded by Hudellet at Dinguirah Mahina Ouh between Kayes and the Niger in the Sudan while cases

According to Seidelin it is a small protozoon (*Paraplasma flavigenum* Seidelin 1909) found in the red blood cells but the parasitic forms described by this author are now considered to be artefacts

It is obviously a living organism and not a chemical substance because of the time it takes to develop in man and the mosquito for the incubation period in man is usually three days The mosquito in order to become infected must bite a patient during the first three days of his illness and then four or five days must elapse before the infected mosquito can transmit the disease

The proof of the transmission of the disease by *Stegomyia calopus* was worked out by Reed Carroll Agramonte and Lazear by construction of a great proof building divided into two great proof compartments into the first of which infected mosquitoes were liberated and allowed to bite a non immune while other non immunes slept in the second compartment The man in the first compartment

of yellow fever did not contract the disease though after this experiment was finished some were infected by means of the bites of infected mosquitoes thus proving that they were not immune

Marchoux and Simond extended this knowledge by showing that a *Stegomyia* can live for some thirty days and lay seven batches of eggs Those laid after the twelve days incubation by an infected

the day and during the night

From this it is argued that any *stegomyia* which bites in the daytime can have only immature parasites, and therefore cannot produce an infection

A mosquito can produce yellow fever some fifty seven days after infection which appears to become more virulent the longer it remains in the insect, especially if the air temperature is  $27^{\circ}$  to  $28^{\circ}$  C

■  
a  
f

Thomas in 1907: No other mosquito so far has been proved to carry the infection. It appears also that the development in the mosquito depends to some extent upon the air temperature, as in the case of the malarial parasite

has suggested that the disease may be kept up by animals

characterized by saying that the  
in the blood stream of man

Meigen 1818 (*S. fasciata*)

Fabricius 1805 non O. F. Muller 1764) by the bites of which it can be transmitted to man and the chimpanzee. The blood of the infected man is transmissible only during the first three to four days. *Stegomyia calopus* begins to be infective fourteen days after the

*Spirochæte*—In 1909 Stimson reported the presence of a spirochæte in yellow fever (*S. interrogans* Stimson 1909) but no importance was given to this observation. Recently Noguchi has cultivated from the blood of several cases a spirochæte somewhat

similar to *S. icterohæmorrhagicæ* and has named it *Leptospira icteroides*.

**Pathology**—In yellow fever the cells of the liver swell and pressing upon the bile capillaries obstruct the flow of bile and cause a hepatogenous jaundice characterized by yellow staining of the skin and tissues and by the presence of bile in the urine. Further the swelling blocks the intralobular capillaries causing

plage which shows its effect on the intestine and spleen

Th 1 - 11 1 - -

occlusion of vessels

An attack generally confers a lasting immunity upon a person and second attacks are rare and it appears that the so-called racial immunity of people living in endemic regions is really an acquired immunity due to mild attacks in childhood. Relative immunity can be acquired by inoculation of infected blood heated to 55° C for five minutes. Further it is found that the serum of a convalescent has some protective power.

**The Blood**—There is no marked alteration in the numbers or

when present being largely caused by polymorphonuclear leucocytes. The coagulation of the blood is diminished and ammonia is thought to be present in mild cases.

**The Urin**—Albumen appears early—as a rule on the second day—and increases in quantity remarkably especially in severe cases. During convalescence it may disappear or may last for months.

cells may

present—f

is dim in l

absent in uncomplicated case

1 -

*The Vomit*—The vomit is often distinguished as white red black. The white is acid colourless or bile-stained and is composed

but this is not constant. The cells of the tubules show fatty deposits. The suprarenals are enlarged.

blood in the cavity. The meninges of the brain are congested and hæmorrhagic spots may be seen.

*Symptomatology—Incubation*—An exact knowledge of the length of the incubation period is of the utmost importance from a prophylactic point of view. Calculated from experimental mosquito

period as generally given

by a remission or intermission. The first attack is characterized

by headache pale then flushed face injected eyes and pains in the body and after the second day albuminuria while the second attack shows the jaundice from which the disease obtains its name and the hemorrhages of which black vomit and black motions form

"

.

the temperature either dropping with a crisis accompanied by

"

at this feature called Raget sign now shows itself in that the pulse does not increase in rapidity as the temperature rises On the

—

and prostration The tongue is dry and furred with red tip and

restlessness and often delirium

Terminations — Two cases

— — — — — The 6 th +



*The Vomit*—The vomit is often distinguished as white, red, and black. The white is acid, colourless or bile stained, and is composed

.. .

.. .

signs of some congestion. Bowman's capsules are said to be dilated but this is not constant. The cells of the tubules show fatty degeneration, and the lumen may contain granular debris. The suprarenal capsules may be hyperæmic or show fatty degeneration but neither of these is constant and hyperæmia or fatty degeneration of the pituitary body and the thyroid gland have been described, but are not important. The bladder is usually empty. The heart

hæmorrhagic spots may be seen

**Symptomatology—Incubation**—An exact knowledge of the length of the incubation period is of the utmost importance from a prophylactic point of view. Calculated from experimental mosquito

two days twenty two hours to six days two hours. Marchoux

The average time appears to be about five days but to be on the safe side at least six to seven days must be allowed

*The Fever*—The fever is divisible into two paroxysms separated by a remission or intermission. The first attack is characterized

by headache pale then flushed face injected eyes and pains in such ominous signs

the temperature either dropping with a crisis accompanied by

characteristic feature called Lager's sign now shows itself in that the pulse does not increase in rapidity as the temperature rises. On the

a high temperature

*Second Attack* --During the second attack all the symptoms of

urine increase in amount the albumen diminish and the vomiting gradually cease while sweating may occur and the patient passing into a deep sleep awakens on the road to convalescence. The second



**Treatment** —There is no specific remedy known for yellow fever. The usual treatment, if the patient is seen on the first or second day is to give repeated small doses of calomel until 35 grains have been administered and then a draught of magnesium sulphate and sodium sulphate. Then employ Sternberg's mixture which is —

Sodium bicarbonate	150 grains
Mercuric chloride	1 grain.
Water	60 ounces

This is given in three tablespoonful doses every hour.

**General Treatment** —As the cause of the disease is unknown the treatment must aim at the prevention of the spread of the infection, the rapid elimination of the toxins and the alleviation of the symptoms. To prevent the spread of the disease the patient's bed room must be rendered gas proof. The excretion of the toxins must take place by the bowels, skin and kidneys and therefore it is necessary to encourage the action of all three. The action of the bowels can be maintained by enemata of 1 tablespoonful of sodium sulphate dissolved in 1 pint of warm water and given

ill can appreciate how embarrassing the weight of heavy blankets is to the heart. It is hoped that by these means the action of the skin will be stimulated.

th  
flu  
4  
four hours or the same quantity of any alkaline mineral water to each pint of which 30 grains of sodium bicarbonate have been added or by Sternberg's mixture as recommended above or an effervescent drink can be made up of sodium bicarbonate and fresh lime juice but care should be taken that this is mixed in such quantities as to be neutral and it may if necessary be combined with sodium sulphate. If this alkaline treatment cannot be given by the mouth then the bicarbonate of soda must be administered as warm enemata.

**Symptomatic Treatment** —As regards symptoms if there is great pain this can be relieved by a small dose of phenacetin while severe lumbar pain may be relieved by hot fomentations but if no urine is passing the warm mustard bath will be better.

Vomiting is treated by sips of iced Vichy iced champagne a blister to the pit of the stomach a dose of a mixture containing chloroform or a hypodermic injection of morphia. If there is much fever this must be treated by cool sponging and cool applications to the head.

Black vomit should be treated by the application of the ice bag

to the pit of the stomach, and hæmorrhages in general should be met with doses of calcium chloride or hypodermic injections of adrenalin or ergotin

In these bad cases it is advised by some authors to rub olive oil into the skin in quantity, with a view of loading the endothelial cells of the blood capillaries with fatty particles, and so protecting them against the action of the toxins

Anuria requires treatment by hot fomentations to the loins, or

Cardiac failure requires hypodermic injections of strychnine, camphor dissolved in ether or oil, or simply ether

*Diet*—No food should be given for two or three days but only the alkaline drinks, and champagne if necessary the idea being to relieve the stomach and prevent the accumulation of waste products in the system and so to save the kidney from too much work

After this time or if the temperature is below 102° F milk and lime water, toast water and barley water may be given With a view to saving tissue waste and getting fat into the circulation, pure frozen cream cold white wine-jelly or lemon jelly, should be given, or olive oil administered by the mouth or rubbed into the skin as indicated above

After the temperature has been normal for some three days chicken broth, custard pudding, blanc mange etc can be tried, and a few days later the diet can be gradually increased

Strong beef extracts and strong alcoholic stimulants should be avoided, the best stimulant being iced champagne in tablespoonful doses

**Prophylaxis.**—Yellow fever is generally conveyed from one place to another by ships, but in order to produce an epidemic several factors are necessary—viz, cases of the disease from which *Stegomyia calopus* may become infected, together with conditions of temperature moisture, etc, suitable for the development of the germ in the mosquito, and also for the propagation of the mosquito itself, together with the presence of non immune people for the mosquito to infect It is also necessary to bear in mind that the incubation period is usually from three to six days, and therefore quarantine must be of at least five days or to be on the safe side, of six or seven days duration In order that the mosquito may become infected it must bite a patient during the first three days of the illness, and another fourteen days must elapse before it is able to infect non-immunes Therefore an infected

the shore  
 ■ proof  
 and the  
 whole ship disinfected by a Clayton's disinfecter, preferably between 9 a m and 3 p m, while the mosquitoes are quiescent.

*Sicromyia catopis* is essentially domestic in its habits, that it is active from 2 p m till early morning, but that it is quiescent between the hours of 9 a m and 2 p m, when, therefore visits can be made to infected areas without risk. Further, the mosquito is known to bite dead bodies and suck the blood but this will seldom infect it, as a patient generally lives longer than the three days during which the disease can be communicated to the mosquito. Lastly, it can pass through a screen with fifteen meshes, but not through one with twenty meshes to the inch.

The mosquito does not die after laying its eggs but lives until it has laid seven batches—i.e., some thirty days. The eggs laid twelve days after infection are capable of carrying the infection into the second generation, which can spread the disease fourteen days after becoming imagoes. As the mosquito is believed to be non-infective when it bites in the daytime, non-immunes may visit an endemic area in the day with impunity, but must not stay late in the afternoon.

When a person is moved from an infected room, disinfection should be begun at once. All cracks openings etc., should be closed with paper, and fumigation carried out, preferably by means of sulphur dioxide gas, or if there is an objection to this because of the damage it causes, pyrethrum may be used, but must be burned in the proportion of 1 pound to 1,000 cubic feet of air-space if the mosquitoes are to be merely stupefied and 2 pounds if they are to be killed, or tobacco 1 pound per 1,000 cubic feet, may be used.

In addition, if an epidemic is to be eradicated cases must be at once notified to the central authority, and patients must be strictly treated in mosquito proof rooms, and every person, immune or non-immune, must use mosquito curtains, while an anti-mosquito scheme on the lines mentioned under Malaria must be undertaken. Special care must, however, be taken to eradicate, after a careful

old tins  
is, houses  
ly when  
granted  
that the importation took place at least two to three weeks prior to the discovery, for this is the time required for the incubation of

must be won over to assist, and offenders must be fined for transgressions against sanitation, as is done by the Americans in Panama,

the offences being the presence of mosquito larvæ, imperfect screening of the house, accumulation of water, etc.

The prophylactic measures may be summarized as follows —

*A Where there is reason to believe that yellow fever is endemic*

- (1) Segregation of non-immunes partial or complete town planning
- (2) Screening —
  - (a) The bed
  - (b) The veranda
  - (c) The house
- (3) Systematic mosquito destruction —
  - (a) Removal of breeding places
  - (b) Screening of water cisterns
  - (c) Oiling
  - (d) Drainage

(4)  
(5)  
(6)

*B Where yellow fever has broken out*

- (1) Removal of all non-immunes outside the infected area and deflection of the traffic outside the infected area
- (2) Isolation of cases and suspected cases
- (3) Provision for isolation of contacts
- (4) Early notification
- (5) Fumigation
- (6) Emergency mosquito measures —
  - (a) Removal of receptacles
  - (b) Oiling
  - (c) Screening
  - (d) Drainage
- (7) Education —
  - (a) Lectures
  - (b) Meetings
  - (c) Pamphlets
- (8) General organization of the medical forces

No anxiety need now be felt as to the spread of yellow fever to Asia though the Panama Canal has been opened for it appears as though not merely yellow fever but all infectious and contagious fevers are bound to be eradicated from the Canal zone under the able American rule

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## CHAPTER XLIII

# DENGUE AND ALLIED FEVERS

Dengue—Seven days fever—Dengue like fevers—References

### DENGUE

**Synonyms**—*Febris Endemica cum Roseola Exanthesis Athrosia* Knok  
kelkoorts *Arthrodynæ* *Fièvre Rouge* and probably the seven days fever

There are upwards of one hundred known synonyms for this fever many of which refer to it as a form of rheumatism or give it fanciful terms such as breakbone or breakheart broken wing or giraffe fever. It is some times called three days fever a term by which pappataci fever is also known.

The word dengue is said by Vambery to be of Arabic origin and to mean weakness but it might equally be of East African Indian or Spanish origin.

**Definition**—Dengue is a term covering one or more acute specific non contagious fevers of unknown origin but caused by a virus contained in the blood. It is characterized by a high fever, aching joints, and a skin eruption.

agency of *Stegomyia calopus* Meigen 1818 and probably by *Culex fatigans* Wiedemann 1828

**History**—It appears to have been first discovered in Java in the year 1779 by Pazzio previous to this it was soon discovered in other places being found from 1779 to 1780 in Egypt Arabia Persia and North America and in 1784 in Spain to which it appears to have come from the West Indies and in 1818 in Peru. In 1824-28 it occurred all over the tropical and subtropical zones and in 1830-70 it formed a series of large and small epidemics in the same regions.

From 1871-73 there was an epidemic in Egypt Arabia India Burma later to America and North America all the tropical regions. In 1877-80 it occurred in Egypt Syria and the islands in 1885 to which it was introduced from the complaint. In 1889 it was especially prevalent in Asia Minor Turkey and Greece.

From 1890-93 it was especially prevalent in the East Indies and in 1894 it was especially prevalent in the East Indies and in 1895 it was especially prevalent in the East Indies.

1897-98 it was epidemic in Georgia Florida and Texas In 1901 it occurred in Penang and Asia Minor in 1902 in Hong Kong, Singapore Madras Rangoon and Upper Burma In 1906 it occurred in Saigon and in France and in 1907 it has been

first made by Pazzio and Bylon and the first full clinical description given by Dickson in 1828

The first attempt to find a causation for the disease was by McLoughlin in 1836 who found micrococci in the blood but this was disproved by Klein Wright Crookshank and Macfadyen In 1903 Graham stated that he had observed small hyaline unstainable rods or dots in the red blood corpuscles which he considered to be protozoal parasites of the nature of Plasmodia This discovery

the fifth day after feeding He did not find any oocysts or oocysts but he says he found spores in the cells of the salivary glands of the mosquitoes from forty eight hours to one month after they had fed on infected blood Further he states that he caused a typical attack of dengue by injecting subcutaneously a solution in normal saline of the salivary glands of a mosquito which had bitten a dengue fever patient twenty four hours previously He only performed one experiment because the illness caused was very severe but he succeeded after an incubation of four to six days in infecting healthy people by the bites of mosquitoes (*Culex fatigans*) fed on dengue fever patients

Though Ashburn and Craig (in 1907) have not supported Graham by finding the parasite they have supported him by proving that a typical attack of dengue can be produced by the intravenous inoculation of filtered and unfiltered blood from an infected patient and that the disease can be and usually is transmitted by the bite of *Culex fatigans* Wiedemann

In 1912 and 1913 Lalour drew attention to *Stegomyia calopus* as the carrier of dengue Cleland Bradley and McDonald demonstrated that the virus of dengue fever was carried by *Stegomyia calopus* in which it underwent development Archibald found that the same insect was the only possible carrier in certain parts of the Sudan These observations support Legendre's earlier views

the sub tropical zones Cases have been reported from Southern Europe Its usual boundaries are 32° 47' N and 23° 23' S but during warm weather it may spread to 36° 10' N and even to

42° N and 28° S in exceptional cases. It is, therefore, a disease of tropical climates, and of warm weather in other climates. It appears to be favoured by low-lying lands near the sea, well supplied with water, and not to be found at high altitudes; but there are exceptions to this. It occurs in Australia, where it has appeared since 1885, and is common in the Anglo-Egyptian Sudan and along the Red Sea.

When the distribution of dengue is compared with that known for *Culex fatigans*, it will be seen that the two coincide most re-

#### ætiology of this disease

There appears to be no doubt that dengue fever is caused by some unknown living organism which requires over two days to increase so as to produce the symptoms of the disease when inoculated into human beings, and generally five to nine and a half days when produced by bites of an infected stegomyia.

It is so small that it will pass through the pores of a filter which will retain *Micrococcus melitensis*, which is only 0.4  $\mu$  in diameter. It is intratypical, and the incubation period is three to five days.

lymphatic glands the gland juice being at times infective, as proved by experiments.

by an attack

The points in the ætiology which require further research are the

**Pathology** —There is but little that can be written on this subject as the cause of the disease is unknown and therefore of course its

in the case of *culx* and five to nine and a half days in the case of *stegomyia*. Prodromata are usually absent but general malaise may be felt during a period not exceeding three days before the onset.

The *attack* is usually sudden and introduced by severe pain in some part of the body or merely by the sensation of extreme fatigue or by chilliness and shivering or by deep flushing of the face. In children convulsions or delirium may usher in the illness. In any case the temperature rapidly rises, the pulse quickens and the skin and mucosæ become markedly congested especially about the face, mouth and throat. The conjunctivæ are injected, the eyes watery and sometimes vomiting and purging are also present. The disease now progresses for one, two or three days after which there is as a rule a remission or an intermission.

**Course** —During this period the temperature rises to  $103^{\circ}$  to  $106^{\circ}$  F and the pulse increases proportionately with the temperature to 90 to 120 per minute. Severe pains in the head and

of these glands has reproduced the disease in two out of three cases. The circulatory system is not affected except that fainting occurs at times while a sensation of præcordial discomfort or of suffocation may be complained of. Insomnia and delirium are not infrequent but meningitis and neuritis must be uncommon if they occur at all though hyperæsthesia of the skin may be observed. The skin shows the congestion already mentioned which is not always present.

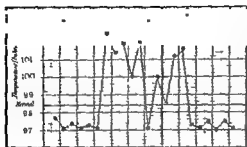


FIG 630—TYPICAL TEMPERATURE CHART OF DENGUE FEVER

quite normal except that it is high coloured but may contain a minute trace of albumen and in severe cases Guiteras says the diuizo reaction can be obtained

**Intermission**—On the third day the temperature usually falls but rarely reaches to normal. This fall may be accompanied by the usual signs of a crisis—namely profuse perspiration, the passage of much urine, violent diarrhoea and bleeding from the nose—but in many cases these signs may be absent. The patient now feels better, except for slight pains, and a genuine intermission in the disease is established. In other cases however this intermission may be entirely wanting and the temperature may only remit remaining at  $100^{\circ}$  to  $102^{\circ}$  F.

**Second Attack**—The intermission or—generally—remission lasts until the fifth day of the illness when the temperature rises to some point usually below  $103^{\circ}$  F, and at the same time a rash appears on the palms and backs of the hands and rapidly spreads to the arms, the trunk, and the legs. This rash may be of two types—a measles eruption of small circular dark red maculo-papules, which almost entirely disappear on pressure, or a scarlatiniform eruption of close set bright red points which may coalesce and form large red patches. Eruptions in intermediate appearance between these two types may, however, be present, and occasionally only an abortive or no eruption at all occurs.

Leucopenia is so constant as to be of diagnostic importance, varying from 4,860 to 1,200, the average being 3,800 per cubic millimetre. The leucocytes are normal in appearance, but there is an increase of the large mononuclears and eosinophiles and a decrease in the polymorphonuclear leucocytes. The urine appears to be usually

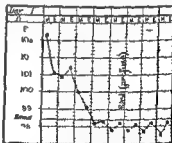


FIG 631—THE ATYPICAL TEMPERATURE CHART OF DENGUE FEVER

continue for two to three weeks.

The typical disease shows a first attack, a remission or intermission, and a second attack, though the two attacks may become merged together. The typical rash may be absent, and sometimes the disease is so mild that it ends on the third day with the first crisis.

Convalescence may be quick and permanent, but, on the other hand, it may be protracted and complicated with sequelæ. Immunity is said to be complete twenty-four days after recovery from a typical attack.

Varieties —It is now generally agreed that seven days fever is

and corresponds with one variety of the old *jeopichia*, and cannot

the mucosæ, as already mentioned, of the nose, stomach, intestines,

appendicitis in two cases.

Sequelæ —The most important sequelæ are the pains in the joints and muscles which worry patients considerably. This polyarthritides affects some of all the joints.

**Reinfections.**—These are not uncommon, and each attack may be quite typical, being associated with the rash

**Diagnosis.**—This is based on the sudden onset with extremely severe muscular pain, the remission or intermission in the course of the fever on the third or fourth day, the rash generally appearing on the third to the sixth day. The diseases most likely to be mistaken

by the absence, as a rule, of any eruptions and the presence of catarrhal symptoms *scarlet fever* by the presence of the sore throat with enlarged cervical glands, *measles* by the catarrhal symptoms and the absence of the severe pains, *rheumatic fever* by the swelling of the joints, *smallpox* may be with difficulty recognized until the eruption comes out, and *tonsillitis* may be recognized by examining the throat. In the tropics, often begins suddenly, there are, however, rare in enteric, and the course of the fever will clear

day

**Prognosis.**—This is quite good as the mortality is usually nil, but in Australia it caused 1 death in 1,000 cases principally in those under five and over sixty years of age

**Treatment.**—No rational treatment can be given as we do not know what the nature of the cause will prove to be. Symptoms must, however, be relieved. The fever and headache may be com

be administered  
tomel. The diet

**Prophylaxis.**—Protection against mosquitoes as described under Malaria, excluding, of course, quinine prophylaxis, is the correct method of preventing the disease

### SEVEN DAYS' FEVER.

**Remarks.**—In our opinion the seven days fever is dengue or a variety of it. We have come to this conclusion after having had the opportunity of studying epidemics of dengue and of the so called seven days fever in various countries

**History.**—This disease was described by Rogers in 1905-08 as a sporadic fever of Indian seaport towns. It is probably identical with Crombie's simple continued fever, and according to many observers including ourselves is a form of dengue.

may be caught by mosquitoes. The disease is carried by mosquitoes. The fever is commonly found among Europeans and especially among people having to do with shipping and also in new-comers.

**Pathology.**—The morbid anatomy is unknown as the mortality is nil.

**Symptomatology.**—The incubation period is unknown and no prodromal symptoms have been recorded. The invasion is sudden with a rapid rise of temperature to 102° or 103° F but the pulse is not as quick as it should be compared with the temperature.

The face is flushed and the palpebral conjunctivæ injected and some rose coloured spots may appear on the skin. Pain is felt in the back and less commonly in the limbs. After the initial rise there is usually a gradual marked remission of the temperature for two to three days when the fever again rises producing the typical saddle back remission. After the second rise the tem-

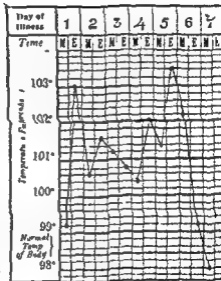


FIG 632.—TEMPERATURE CHART OF A CASE OF SEVEN DAYS FEVER

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vomiting  
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Rashes are frequent. The most common is a mottling or a diffuse erythema of the extensor surfaces of the forearms on the fourth to sixth day which



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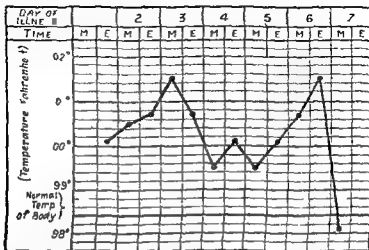


FIG 633.—TEMPERATURE CHART OF A CASE OF SEVEN DAYS FEVER

**Diagnosis**—It can be distinguished from malaria by the absence of parasites and the slow pulse with high fever from influenza by the absence of respiratory symptoms

**Treatment**—This is purely symptomatic

### DENGUE-LIKE FEVERS

#### VAN DER SCHEER'S FEVER

**Synonym**—Five days fever

**Definition**—A fever usually lasting for five days and associated with an eruption of red macules and papules about the size of a pin's head occurring on the trunk. It may be a form of dengue

abdomen. After five days remittent fever the crisis comes on the sixth or seventh day when the temperature rapidly falls below normal. This fall is accompanied by moderate sweating. In more severe cases the crisis is absent and cardiac failure setting in the patient dies cyanosed.

that injections of caffeine be administered on the sixth day to prevent a possible attack of cardiac failure

### IM-PYENG

According to Lands and Maignon there exists in Corea a fever called by the inhabitants Im Pyeng. It is most frequently met with in the country districts from February or March to July when it is most common among the poor but affects all classes of society. It would appear to us to be all ed to dengue fever.

decline being associated with a profuse perspiration

Sequelæ.—The patient is left with both general and cardiac weakness after an attack

Treatment.—Quinine sulphate in association with ant pyrin is the treatment usually adopted

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## CHAPTER XLIV

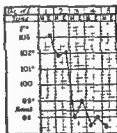
### PAPPATACI FEVER

Synonyms—Definition—History—Climatology—Etiology—Pathology—  
 Morbid anatomy—Symptomatology—Diagnosis—Prognosis—Treatment  
 —Prophylaxis—References

**Synonyms.**—Three days fever Phlebotomus fever Sandfly fever Summer  
 fever *French* Fièvre des trois jours Fièvre de Pick Fièvre de Pym  
 . . . . . Influenza estiva  
 . . . . . *German.* Hund-  
 . . . . . in Febricula pro

as occurring in the Mediterranean basin and this was confirmed by Burnett in 1816. From that time constant references may be found to this complaint in the English Army reports concerning Malta, where it was sometimes called 'summer fever'. In 1855-56 there was a considerable number of cases in that island, due to the passage of the troops *en route* to the Crimea. The fever was also described by Cicoli in 1874-75 in Pola, by Pick in 1887, by Karlinski in 1889, by Taussig who was the first to suspect *P. papatasi*, in 1905, and in the same year by Panec. In 1903 McCarrison was the first observer to differentiate the disease, which he did in Chitral, when he suspected the sandfly as the causal agent. In 1907 it was noticed that although undulant fever had almost disappeared from Malta there were no less than 340 admissions for 'simple continued fever' into the Army hospitals, and in this year Gerrard and Marratt drew attention to this fever. In 1909 Doerr,

In temperate climates, it only occurs in the summer months, and, when studied epidemiologically, it is found to be correlated with the distribution of *Phlebotomus papatasi*. How the infection is maintained during the winter months is not understood, as the imagines do not live through the winter, and as a relapse after a long period is unknown. Doerr believes that the female flies transmit the infection to their progeny, which in turn restart the disease during the



FIGS. 634 AND 635.—TEMPERATURE CHARTS OF PAPPATACI FEVER

Scopoli 1786 is the carrier of the disease but does not become infective at once after feeding, but, on the contrary, is not infective under a week after which it can convey the infection. This proves that the organism...

experimental cases varied from three days sixteen hours to seven days, and a few of these cases showed only the gastro intestinal symptoms without any sign of fever. Animals have so far not

Pathology. - Modules appear to be generated during an attack, as Doerr has shown that the serum from convalescents may neutral-

ize infective serum moreover this action may be demonstrated

endemic region are believed to be immune possibly because there has been a previous attack during childhood

**Morbid Anatomy**—In the few post mortems which have been performed the signs exhibited have been those of the complication from which the patient died as the disease *per se* is not fatal

to  
pa  
but rare in Europe and Africa.

**Attack**—The onset is sudden with a slight rigor or a feeling of chilliness and severe frontal headache lumbar and body pains

from The skin is hot and dry and the temperature rapidly rises reaching 104° F or rarely 105° F in twenty four hours The pulse may reach 100 to 116 but is often unduly slow The patient is very irritable and intolerant to sounds Movements of the eyes are painful the conjunctivæ are injected and often show a red band running from the cornea across the sclera Sleep is impossible at times but at other times the patient may be very drowsy The tongue is coated on the dorsum with a white or brown fur and the edges may be red The appetite is lost and the sense of taste destroyed but vomiting is uncommon though there is pain in the epigastrium and sometimes diarrhœa The mouth and throat are congested and irritable and the gums may show a tendency to bleed and there may be a little bronchitis The cough is generally dry with thick tenacious mucopurulent expectoration The tonsils may be enlarged and the uvula congested but this is not constant Not only is there the congestion of the tonsils and pharynx but the mucosa of the soft palate presents a peculiar appearance showing small hyperæmic roundish spots The eruption is sharply limited by a line of demarcation between the soft and hard palates This appearance is not peculiar to the disease

sensations in the palms and soles Cramps are not uncommon Irritation is frequent and delirium occasionally seen



slowness of the pulse and the leucopenia but both diseases may exist together from sunstroke by the absence of the severe symptoms nervous symptoms and the lower temperature from dengue fever it can only be distinguished by the fever ending the third day and by the absence of the rash In countries where pappatagi fever and typhus are endemic the diagnosis at the onset between these two fevers may be extremely difficult Examine the blood in pappatagi there is generally leucopenia in typhus no leucopenia frequently leucocytosis

**Varieties**—An afebrile variety with only headache and body pains is described as well as an abortive form lasting two days Relapses and true reinfections may also occur

**Complications**—The complications are bronchitis and phlebitis

**Sequelæ**—Pains in the bones neuritis and a peculiar loss of memory may be sequelæ

**Prognosis**—This is good as no one has been known to die of the uncomplicated complaint

**Treatment**—The treatment is purely symptomatic and consists in sending the patient to bed in administering a saline purga

Franz and Kolar recommend the subcutaneous or intravenous injections of colloidal silver but this hardly appears necessary in such a mild fever Atoxyl has been found to be useless and quinine to be harmful

During convalescence a change of air and an iron tonic may be recommended

**Prophylaxis**—The only obvious means of prophylaxis consists in isolating the sick and protecting them against the phlebotomus by means of mosquito curtains of a sufficiently fine mesh It must be remembered that the little fly bites mostly in darkness and chiefly in houses

As regards ordinary prophylaxis a fine mosquito curtain together with the use of camphor is to be tried Fumigation by burning pyrethrum may also be tried

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## CHAPTER XLV

# THE AFRICAN TRYPANOSOMIASES

General remarks—The sleeping sicknesses—The trypanosome fevers—  
References

### GENERAL REMARKS.

THE human African trypanosomiases include two different clinical conditions—viz —

A The *sleeping sicknesses* characterized by the fact that we definitely know that the preliminary fever leads to meningo-encephalitis and meningo myelitis

B The *trypanosome fevers* characterized by the fact that we do not know that in these cases the disease will end in meningo-encephalitis and meningo myelitis

Before Castellani discovered a trypanosome in sleeping sickness, Ford and Dutton had demonstrated that 'Gambia fever' was due to a trypanosome. After this discovery this disease was named 'trypanosome fever,' and was considered to be quite distinct from sleeping sickness. After Castellani had found a trypanosome in sleeping sickness the two diseases, trypanosome fever and sleeping sickness, were at the time judged to be covered by the one name 'sleeping sickness. Recently, however, *Lanfranchi's laboratory infection* with a trypanosome has lasted for more than seven years, and there is no sign of meningo encephalitis, as may be judged *inter alia* by the excellent papers which the distinguished professor produces. In fact, judging by the symptoms exhibited by Lanfranchi which resemble those found in the trypanosomiases of animals, it seems very doubtful whether he will ever show signs of what we clinically call 'sleeping sickness.' Further, the organism with which he is affected appears to be of the *Evans' type*, a form known to be in his laboratory when infection took place.

Under these circumstances, it appears to us to be useful to return to the old name *trypanosome fever* for infections of man and animals



## THE SLEEPING SICKNESSES.

**Synonyms.**—Sleeping dropsy, Negro lethargy, Morbus dormitivus  
*French* Maladie du Sommeil *Portuguese*: Doença de Sonno *Italian*.  
 Malattia del Sonno, Letargia dei Negri. *German*. Afrikanische Schlaf-  
 krankheit Trypanosomen Fieber. Native names are very numerous—  
 Lafangola Lía La negulo N tansi, Mongota, Konje Marree, Kaodzera N'dula  
 Tula Manugina Nelavare Dadane, Toruahebu, etc

**Definition.**—The sleeping sicknesses are chronic specific fevers

by *Glossina morsitans*, characterized by an inflammatory condition of

have been compelled to introduce new terms which we have made  
 as few as possible by utilizing those found in the literature with  
 which we are acquainted Two of these new terms occur in this  
 chapter, because we have gone more fully into the trypanosomes of  
 man than into those of animals, because this is a work on tropical  
 medicine and not upon trypanosomes, and because those of man  
 are the forms which have been most satisfactorily studied

Chalmers has introduced the name *Castellanella* as the generic

upon which practically all the work in connection with sleeping  
 sickness was done prior to the discovery of *C. rhodesiensis*, which is

or is it realized that  
 morphologically alike,  
 from a pathological  
 and serological point of view as there is a suspicion that the well-  
 known difference in type between the milder form of the disease as  
 seen in portions of the West Coast of Africa and the severer as seen  
 in Uganda may not be due to acquired partial immunity, but to a  
 difference in the two organisms

*C. castellanii* as found in sleeping sickness and *C. gambiense* as  
 signs of sleeping

It appears to us that *Castellanella gambiensis* as seen on the West Coast of Africa and as originally described by Dutton and Todd

**History**—The earliest mention of sleeping sickness, so far discovered is by John Atkins in his little book entitled 'The Navy

of the enlarged glands of the neck the association of which with the disease was so well understood that slave traders would not buy slaves who had enlarged glands

In 1808 Moreau de Jonnés described the disease in negro slaves in the Antilles. In 1849 Clarke on the Gold Coast Davis and Daniell on the Guinea Coast and Ferreira came across it at St Thomas. During the next twenty years a number of observers described sleeping sickness among whom it is important to note that Guerin met with it in 1869 in Martinique in negro slaves who had been imported from Africa. In 1876 Corre gave a good description of the disease as he knew it in Senegal

In 1891 the first case was brought to London and was studied by Sir Stephen Mackenzie and in 1900 two more cases were brought to London this time under the care of Sir Patrick Manson. The morbid anatomy of these cases was carefully studied by Dr Mott

Castellanella in Uganda found a trypan so

by previous observers, the fever and the peculiar tremors, and Christy published concerning the disease. I that the trypanosome Desvoidy, a conclusion already reached by Sambon and Brumpt on epidemiological grounds. From 1903-05 much clinical experi-

Medicine Kleine, in a series of important researches, has experimentally shown that *C. castellani* undergoes a cycle of development in *G. palpalis*—a fact which has been fully confirmed and extended by Sir David and Lady Bruce, Hamerton and Mackie, and Miss Robertson, as well as Fraser and Duke.

Koch, Laveran, Mesnil Minchin, Blanchard Greig Gray, Tulloch, Kinghorn, Montgomery, Martin, Pittaluga, Lebœuf and Roubaud, have all studied the disease and its epidemiology, and an International Conference was held in 1907 in London, and a Bureau for the study of the disease founded. This bureau for some time issued monthly bulletins, which are most valuable to the student of the disease but recently it has become converted into the Bureau of Tropical Diseases.

In 1910 Stephens and Fantham created a new species of trypanosome (*C. rhodesiensis* Stephens and Fantham 1910) for the parasites found in cases of sleeping sickness in the Luangeva Valley in Rhodesia, because the trophonucleus of a certain percentage of short forms was situated either close to, or even on the flagellar side of, the kinetonucleus. In 1912 Kinghorn and Yorke showed that this trypanosome was transmissible by *G. morsitans* Westwood, 1850, and in the same year these observers pointed out the importance of the meteorological conditions on the development of the trypanosome in the fly. Further work has been done by Sanderson Murray Shircore, and others. As regards the history of the treatment, arsenic was long ago considered beneficial for the trypanosomiasis of animals, Livingstone being the first to apply the drug to a horse for the purpose of treating nagana. Since then it has been used for the same purpose by several persons, notably by Lingard (1893) for surra and by Bruce (1896) for nagana, while Laveran and Mesnil introduced sodium arseniate in 1902 for the same disease, E. J. Moore and Chichester advocated the use of hypodermic injections of arsenic, and Thomas and Breml of the same of sodium arseniate. In the meanwhile Manson had treated several cases of sleeping sickness with arsenic (liquor arsenicalis), and Ehrlich and Shiga had treated various experimental trypanosomiasis with colouring compounds belonging to the benzo purpurin group of which trypan red is the best known. Laveran and Mesnil also did some valuable researches on the subject. Thomas, in 1905, first brought the drug 'atoxyl' to the notice of the profession as a means of treatment of experimental

trypanosome affections and Kopke in 1906 tried it in human beings affected with sleeping sickness.

The beneficial action of atoxyl in sleeping sickness was further confirmed by Broden van Campenhout, Minson, Koch and many others. In 1917 Ehrlich and his pupils Franke and Roehl dis-

covered an emetic which has been found to be very beneficial especially if

administered in small doses. This fact clearly proved that though the disease may be introduced by man along

the coast and at the Stanley

Stanley had travelled  
with his followers and had  
delays on the Victoria  
with some of Stanley's  
about Kivali to the

west of the Albert Nyanza were brought by Sir F. Lugard for  
political reasons to Busoga and Uganda. Christy and Hodges

long it would pass from the Congo into Eastern Rhodesia. Since  
then sleeping sickness has been found in that country though  
the trypanosome found in Rhodesia being a different species it is  
more probable that the disease has been endemic there and not

recognized for a long time towards from Uganda for G Victoria Nile but not as earlier than 1908 and in 1909 the disease was recognized in the Bahr el Ghazal Province of the Sudan

Therefore the distribution of the disease at the present time may

discovery of the tsetse fly in Arabia makes it possible that the disease may some day still farther extend its area

There does not appear to be any seasonal influence

**Ætiology**—From the present state of our knowledge it would



FIG 638—TRYPANOSOMA IN CEREbro SPINAL FLUID

Preparation from the cerebro spinal fluid of a case of sleeping sickness



FIG 638A LYMPHOCYTIC ACCUMULATION AROUND A VESSEL IN THE BRAIN

appear that from a clinical and ætiological point of view there are three types of sleeping sickness which may be differentiated as follows—

- I *The Equatorial type* caused by *Castellanella castellani* and spread by *Glossina palpalis*
- II *The Southern type* caused by *Castellanella rhodesiensis* and spread by *Glossina morsitans*
- III

For description of the parasites see p. 417

Ducloux's researches made it possible that there is a vertebrate reservoir for *C. castellani* in the antelope because he found a trypanosome very like this organism in these animals and because

some boys working on an uninhabited island in Lake Victoria

water hogs and domestic dogs but with the human crossed immunity and serological experiments detailed on page 476 it again becomes evident that these two trypanosomes are distinct and therefore the question as to the existence of *C. rhodesiensis* in lice. The con- morphological

we have con sidered the tsetse in relationship to its carriage of these organisms and in Chapter XXIII p 837 we have described these flies *C. castellani* is spread by the agency of *Glossina palpalis* and *C. rhodesiensis* by *Glossina morsitans* but it is not proved by actual experiment that *A. curvicauda* *C. curvicauda* *T. curvicauda* *U. curvicauda*

but there is no evidence that the infection is passed on to the suc

for the lymphatic tissues of the intestine (solitary or agminated

and in a lymphocytic accumulation around the vessels. These two processes compress the vessels and lessen the supply of blood to the cells of the brain and spinal cord in which as the result of malnutrition changes ensue which produce the typical symptoms of the cerebral stage of the disease which is often called sleeping sickness. The trypanosome apparently cannot pass through the placenta as infected women give birth to healthy babies.

Towards the end secondary infections with bacteria may take

researches on the leucocytic formula that he discovered trypanosomes for the first time in sleeping sickness. More recent researches are those by Broden and Rodham and others. During the first stage of the disease it is usually of normal appearance and clear while on centrifugalization there is practically no sediment though occasionally a few small mononuclear cells may be present and trypanosomes are as a rule absent.

In the sleeping sickness stage it is often slightly turbid and contains an amount of serum albumen and serum globulin and on centrifugalization some sediment is obtained consisting of a few like and present

chromatin masses as described by one of us in 1913. These may probably be compared to the so called latent forms described in

and in the lymphatic glands but pathological changes brought about by complications may also be noted.

The body is usually emaciated and anæmic, rigor mortis is well marked and the skin may be normal or dry and desquamating or may show pustular eruptions on the hands and forearms or ulcers on the feet which are generally due to jiggers (*Dermatophilus penetrans*). Enlargement of the lymphatic glands of the neck and groins is generally easily seen. On opening the brain-case it will be noted that the under surface of the scalp is pale, that the dura mater may or may not be adherent to the bone, that the cerebro-spinal fluid is increased in quantity and the gyri of the brain are often

flattened. On careful examination the pia arachnoid will be found to be thickened in places and may or may not be adherent to the grey matter. The brain substance which is generally firmer than normal but may be soft and œdematous is usually congested and the fluid in the ventricles is increased.

With regard to the spinal cord it will be seen that there is an increase of fluid which if examined with the microscope after centrifugalization generally shows leucocytes and trypanosomes. The cauda equina may be found at times surrounded by gelatinous tissue. The cord itself is often congested and hæmorrhages have been described.

The lymphatic glands of the submaxillary region anterior and posterior triangles around the bronchi of the mesentery behind

straw coloured fluid and the pericardial fluid may also be ex



ing a clear eosinophile area (*d*) a few mononuclear leucocytes (*e*) a few polymorphonuclear leucocytes. The cells of Marschalló are considered by Mott to be derived from lymphocytes and the morula cells to be degenerated cells of Marschalló. Mott considers that the lymphocytes are probably formed by proliferation of the endothelial cells of the perivascular lymphatic space. According to Breinl a layer of blood cells may be found external to the round cells and hæmorrhages may be found in the cord. The ependyma of the lateral ventricle also shows a proliferation and dense fibrous

these changes in the brain and spinal cord of a cured case of trypanosomiasis who died several years later of cystic disease

The histological lesions of the encephalitis found in sleeping sickness closely resemble those of general paralysis and encephalitis lethargica (nona)

The next series of characteristic changes are in the lymphatic glands in which trypanosomes are often found. These changes begin with a conversion of the lymphocytes into cells of Marschalló and these again into morula cells. The endothelial cells of the

n,  
mc  
ue  
of

the lymph sinuses and of the vessels become thickened

A lymph nodule therefore appears to be surrounded by a fine connective meshwork containing few lymphocytes but many red corpuscles and phagocytes. As time goes on the inflammation in

and  
may

even

when normal to the naked eye while the complication of pneumonia will give rise to the usual appearances. The heart shows small celled infiltration in all its layers with sometimes hæmorrhages. Vianna has noted in animals infected with *C. castellani* cysts in the muscles similar to those of *S. cruzi* and appears to have found them also in the muscular and nervous tissues from a case of sleeping sickness. The liver and spleen may show thickening of the capsule while the latter is very congested and its trabecule are increased in thickness. In natives signs of chronic malaria are almost

constantly present in the spleen and liver. The bone-marrow may be very cellular with congested vessels and hemorrhages.

Stenson and others have found trypanosomes scattered through the brain substance in no special relation to the capillaries and smaller bloodvessels in animals inoculated with *Castellanella gambiense* (*n. gambiae*) and other trypanosomes.

**Symptomatology**—The course of the disease may be roughly divided into three stages—the incubation the febrile called also glandular stage and the cerebral stage.

**Incubation**—The duration of the incubation period is not certainly known in man but may be considerably shorter than was believed by the old authors probably in most cases it does not exceed two or three weeks and according to Martin and Lebœuf's observations in Europeans it may be even less than ten days. On the other hand some infected individuals may not show any sign of disease for months and it is said even five or six years. The bite of the infected *Glossina* gives rise as a rule only to a very slight local irritation which quickly subsides and is often overlooked by the patient.

**Febrile or Glandular Stage**—The onset of the disease is characterized by attacks of fever almost constantly associated in Europeans with an erythematous eruption. This fever lasts about a week and disappears to recur again later for the same or much longer periods. It is generally of an intermittent or remittent type. During an attack the pulse rate and the respirations are increased and there is often enlargement of the liver and spleen though how much of this may be due to recurrent malaria is not known. The pulse often remains rapid during the afebrile periods. Neuralgic pains and headache may also be complained of.

An erythematous eruption is often found on Europeans. It begins as a rule with badly defined pinkish patches which clear in the centre until a ring is produced. If a portion of the ring fades



FIG. 639.—NEGRO PATIENT IN THE LATE STAGE OF SLEEPING SICKNESS.

a crescent may be produced. This circinate eruption may appear on any part of the body, but is especially frequent on the trunk,

condition of the skin is found. These various eruptions are called *Trypanides*.

The most t  
of one or mor

e enlarged glands  
soft consistency  
general, intense

deep hyperæsthesia. As repeated attacks of fever increase, the patient may become anæmic and asthenic, but the febrile condition may last for years and indeed in this stage the disease may be cured.



FIG. 640.—SLEEPING SICKNESS, LATE STAGE, IN ANGO-EGYPTIAN SUDAN.  
(Photograph kindly lent by Colonel A. Balfour.)

*The Cerebral Stage (the So-called Sleeping Sickness)*—After the

to sit quietly or to lie down. He also becomes careless in his work.

is not the prominent symptom so often de  
the  
the  
o in

the legs, and even in the abdominal muscles. The tremor of the tongue and hands may be a very early symptom and may be present in the febrile stage. These tremors may occasionally be so severe as to resemble epileptiform convulsions, but at times the patient

shuffles along, but the superficial reflexes are normal, the deep reflexes may be exaggerated and then there is no clonus. There is inco-ordination in some cases and Romberg's sign may be present. As the disease advances the rigidity is generally in the muscles of the neck and legs, the thighs on the other side. The rigidity is generally hyperaesthesia and at times the patient

in many ways thus for a period the temperature

temperature it is generally imperceptible at the wrist. The heart as a rule shows no abnormality. The

appetite is good and may be satisfactory but constipation is occasionally diarrhoea. The tongue is coated with white fur, the faces of the eyes are injected and show the usual paralytic changes. The spleen and liver may be enlarged.

**The Blood**—The exact condition is but little known with other parasites.

With this number of cases or less with a certain number to be noted the cases the

may be seen. The leucocytes are normal in number, as a rule, with an increase of mononuclear cells, while there may be a terminal polymorphonuclear increase before death.

In fresh preparations the red cells are not evenly distributed, nor do they phenom  
Durham  
This  
thack,  
l with

the latter of whom found, by Moore and Wilson's method of testing the alkalinity of the ash, that the acidity of the blood was increased,

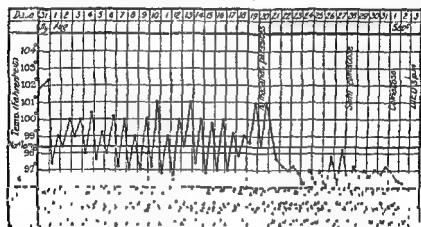


FIG. 641.—TEMPERATURE CHART OF A CASE OF SLEEPING SICKNESS (From Low and Castellani Reports of the Royal Society on Sleeping Sickness)

probably due to the formation of amido-acids either secreted by the parasites or produced by their action upon the proteins of the serum. The acidity was tested by phenolphthalein and Congo red, and the alkalinity, which remained fairly constant was tested by dimethyl amido azo benzol.

The animals were infected with *C. brucei* and *C. equiperdum*, but so far no observations have been made on human blood.

**Urine**—No abnormality is found in the urine but the reaction, amount of phosphates, etc., of course, varies with the food taken. In native patients it is very often alkaline.

smaller and harder. The skin becomes dry and rough but may be perfectly normal though a papulo pustular eruption may at times be noted on the backs of the hands and forearms. Nutrition suffers and the patient generally becomes much emaciated.

As the disease progresses the muscular weakness and emaciation becomes worse and worse the tremors more pronounced the saliva dribbles from the mouth the urine and feces are passed involuntarily and bedsores form while the intelligence becomes more and more affected and the patient passes into a state of coma with a permanently subnormal temperature and an absence of pulse at the wrist and in a short time is liberated from his sufferings by death. The duration of the cerebral or sleeping sickness stage varies from a few weeks to several months. Since the atoxyl treatment has become of general use Hodges has noted that convulsive and mental symptoms are more prominent and that death is often sudden.

Complic  
than try  
Schistosom  
Trichurus  
nalis T  
be found

epilepsy may be observed

Diagnosis—In the first stage (febrile or glandular stage) the disease may be readily confused with malaria and other fevers but in endemic areas the true nature of the malady may be often

(nona) To make a definite diagnosis the demonstration of *C. castellani*, *C. gambiense* or *C. rhodesiense* in the body of the patient is necessary. The following methods should be used—

1 *Microscopical Examination of the Peripheral Blood from the Finger or Ear*—This procedure is often a failure even using thick films.

2 *Scarification of the Erythematous Eruption and Examination of Blood Films*—This method is more useful than the first but may fail.

3 *Nabarro's Method*—Repeated centrifugalization of 10 c.c. of citrated blood and examination of the third sediment. The results are good.

4 *Dutton Todd's Method*—Some citrated blood is centrifugalized in small tubes and the leucocytic layer examined for trypanosomes.

5 *Greig Grey's Method*—Aseptic puncture of the enlarged cervical glands with a sterile syringe. The gland juice is examined for trypanosomes. This from a practical point of view is the most valuable method. To Mott belongs the credit of having first suggested the search for trypanosomes in the lymph glands as a diagnostic method. Balfour has devised a gland holder which is useful in some cases.

6 *Castellani's Method*—Centrifugalization for fifteen minutes of 10 c.c. of cerebro spinal fluid aseptically removed by means of lumbar puncture. The sediment is examined for trypanosomes by making fresh and stained preparations. The result is almost constantly positive in the sleeping sickness stage but negative as a rule in the first stage of the malady.

The technique to perform lumbar puncture is as follows: The patient may

7 *Inoculation of Susceptible Animals*—Ten c.c. or 20 c.c. of blood are aseptically removed from a vein and inoculated into susceptible animals or 10 c.c. of cerebro spinal fluid may be inoculated. The animals most suitable are monkeys, guinea pigs and dogs. The most suitable monkeys according to Throux and d'Anfreville are those of the species *Cercopithecus ruber* while some other species—e.g. *C. fuliginosus*—are almost refractory.

The following biological phenomena and reactions may sometimes be of diagnostic value—

1 *Auto Agglutination*—In many cases of sleeping sickness in wet preparations of blood examined microscopically with a low power the red corpuscles are not evenly distributed nor are they arranged in regular lines but are clumped together—agglutinated—in irregular masses. This appearance is not specific of trypanosomiasis having been found in cases of filariasis, malaria, syphilis and yaws.

2 *Complement Fixation* —Levadits and Mutterlich have applied the Bordet Gengou reaction to trypanosome infections. According to them the complement can be fixed by using as antigen an extract of trypanosomes separated from red cells.

Attempts have been made to evolve agglutination reactions, trypanolytic precipitin and other biological tests for purposes of the diagnosis of sleeping sickness, but so far with little success.

**Prognosis** —The prognosis is serious but not quite so serious as it was before the introduction of atoxyl and the mixed atoxyl-tartar emetic treatment. If the patient is in the early stage and can be removed from the endemic area and put under good hygienic conditions and atoxyl treatment the disease may be cured or at least a marked improvement may be obtained. Great care

patient is compelled to live in the endemic area and cannot be treated with atoxyl the outlook is most hopeless. It is to be noted

caused by it in man runs a more rapid course.

**Treatment** —The only medicaments which have been so far found to be of any real value are first arsenic in the form of atoxyl and in a less degree antimony in the form of tartar emetic, the best method of treatment being a combined treatment with the two drugs.

**Atoxyl** —We recommend 2 to 3 grains of atoxyl by intramuscular injection every third day for at least two years or 0.3 gramme every fourth day for the same period with 0.1 gramme for a child ten years old.

Borden and Rodh

Atoxyl is sodium *p*-aminophenyl arsenate— $\text{NH}_2\text{C}_6\text{H}_4\text{AsO}_2$ —and contains from 25.05 to 20.78 per cent of arsenic according to the amount of water of crystallization. Mono-acetylated atoxyl is  $\text{CH}_3\text{CO}\text{NHC}_6\text{H}_4\text{AsO}_2$

According to Mesnil and Nicolle's observations and the more recent observa



... of Negroes ... the ... these ... to be

trypanosomes. Levaditi and Yamnouchi have also prepared an active derivative of atoxyl which they call trypinotoxyl. Nierenstein thinks that atoxyl is oxydized in the tissues and it is only in the nascent state that it becomes efficacious.

... to the fact that large doses of atoxyl ...

*Combined Therapy*—As the result of the important observations of Ehrlich on the phenomenon of chemo-resistancy which may be acquired by trypanosomes after a long use of the same drug numerous combined treatments have been suggested. Of these, the most important are—(1) antimony and atoxyl, (2) mercury and atoxyl, (3) orpiment and atoxyl, (4) various dyes and atoxyl.

Newham recommend the painless Martindale's injectio antimonii oxida 30 minims (=gr  $\frac{1}{2}$  antim ox) to be given subcutaneously once or twice daily. Apparently the trypanocidal action of antimony is more powerful in the lower animals than in man in whom the results are inferior to those given by atoxyl. A mixed antimony and atoxyl treatment is

Basing his opinions upon the very successful treatment carried out by Captain Sims in R A M C at the Yellow Sleeping Sickness Camp in the Mongalla Province of the Sudan Captain Spence in A M C is treating cases in the Bahr el Ghazal Province as follows —

*A Cases in the early stage —*

- 1 Six intravenous injections each of 6 centigrammes of *arsinoyl* at three day intervals
- 2 Interval one month
- 3 Twelve intramuscular injections each of 30 centigrammes of *atoxyl* at three day intervals
- 4 Interval one month
- 5 and 6 Repeat 1-3
- 7 Three months after last treatment the blood of the patient is inoculated into an animal. If the animal remains uninfected the patient is given a numbered disc and told to report every three months.

Total dosage. *Atoxyl*, 108 grammes in about one year. antimony, 108 grammes.

*B Relapses and cases first seen in a late stage of the disease —*

- 1 Nine intramuscular injections each of 1 gramme of *atoxyl* at ten day intervals
- 2 Interval one month
- 3 Nine intramuscular injections each of 1 gramme of *atoxyl* at twenty day intervals

Total dosage 27 grammes of *atoxyl* in two years

act upon the latent form of the trypanosome while atoxyl would influence the active form. In man this combined treatment has apparently not given any better results than atoxyl alone.

diarrhoea. Throux therefore incorporates in the orpiment pills some opium. Throux's formula is —

Orpiment	20 grammes
Extr opii	0.40 gramme
Gumm	}
Pulv glyceriz	
	q s

To be divided into 200 pills

**Various Dyes and Itoxyl**—Combined treatments of Mesnil's afridol and atoxyl, Ehrlich's paraformin and atoxyl, picroic acid, safranin, trypanavin and other dyes and atoxyl have been suggested, but in man the results have not been so successful as in the lower animals.

**Treatment of Natives**—In the case of native it is necessary to gather them into special sleeping sickness camps in order that treatment may be efficiently carried out. These camps should be in some sterile area and should be provided with a trained medical staff. Patients able to work should be employed to raise crops for their own consumption to supplement the diet provided by the Government.

**■ Symptomatic Treatment**—In addition to the atoxyl treatment or combined treatments malaria and the intestinal parasites must be treated if present. The patient should, if possible, be removed from

animals fails to infect them.

**Prophylaxis**—At the present time prophylaxis must be undertaken on the assumption that the disease is spread from place to place by man along channels of human intercommunication and from man to man by *Glossina palpalis* and *G. morsitans* and that at least in the case of *C. rhodesiensis* there are animals which act as reservoirs of the virus. With regard to these flies further information is required as to their bionomics, though Bagshawe's and Hodges' researches as well as those of Zupitza, Sander, Minchin, Kinghorn and Yorke, Carpenter and others have thrown some light on the subject.

Before enumerating the principal prophylactic measures to be recommended we wish to draw attention to the fact that these as pointed out by Bagshawe will be useless without the co-operation of the natives. This co-operation may be obtained by explaining to them at every possible opportunity the reason for the measure taken. In this missionaries and native chiefs may be of the greatest help. With this proviso we consider the following to be the principal prophylactic measures—

**Public Prophylaxis**—We advocate—

1. The formation of a Central Executive International Board with headquarters in either Paris or London.

2 The formation of an Executive Sleeping Sickness Commission in each political division of Africa in which the disease exists

The different G prevent persons tr into non infected be established fo some authorities t glands should be considered from a practical point of view as trypanosome carriers and prevented from emigrating This is,

the result has not been completely successful as infected flies were found to be plentiful three years after the measures had been carried out The waste land became full of game and wild animals some of which are probable reservoirs of the infection Duke in fact has found *C. castellani* in two marsh antelopes or situtungu (*Tragelaphus spekei*) and believes this observation to be confirmed by the infection of two boys working on an uninhabited island in Lake Victoria

Clearing of the bush along the water's edge for 100 yards and

are liable to be surrounded by tsetse flies which have followed them Indeed some authorities look upon the bath room as a source of European infection

*Destruction of the Animals on which the Fly feeds* —Koch recom

supported the idea of exterminating the big game, because they may be the vertebrate reservoir, but this requires further proof, and is therefore at present too radical a measure

*Destruction of the Pupæ* —  
the jungle fowl to destroy the  
shawe, are found in the turf  
trees Balfour and others  
in various ways Further information, however, is necessary on  
the enemies of the pupæ and adult tsetse flies

*Personal Prophylaxis* —Natives in the fly zones should be encouraged to wear suitable clothing, and the reason explained to them Europeans should be careful not to expose their legs and hands to be bitten High boots, puttees, or leggings should be worn, and where the flies abound gloves and veils, though very uncomfortable, are of service White clothes are better than dark ones, as it has been long observed that the tsetse fly, as well as many other insects, have a preference for black or dark colours The use of volatile substances such as citronella oil has been advised by some Unfor

may be useful in man

*Vaccination* —Attempts at vaccination have not yet entered a practical stage, as inoculation with dead or attenuated viruses have so far failed in the lower animals

#### Summary of Prophylactic Measures

##### I General measures —

- 1 Co operation of various Governments especially in controlling the movements of non infected natives
- 2 Formation of medical posts of inspection at suitable places to prevent infected natives entering non infected areas and *vice versa*
- 3
- 4
- 5

*sub judice* )

##### II Personal measures —

- 1 Avoidance of bites by wearing white clothing high boots puttees and the putty pattern of leggings
- 2 Immediate disinfection of a bite by painting it with tincture of iodine or by applying a solution of formalin (1 in 40)

### TRYPANOSOME FEVERS.

*Definition.*—The trypanosome fevers of man are caused by a monomorphic trypanosome allied to *Duttonella vivax* Ziemann, 1905, and to an unspecified germ allied to *Castellanella exansi*, and

are characterized by milder symptoms and the absence of meningo-encephalitis as far as is known

**Remarks**—In Macfie's case of infection with a trypanosome allied to *D. vivax* apart from slight fever there were no symptoms and after a single injection of atoxyl the trypanosomes disappeared from the blood

In Lanfranchi's case of accidental laboratory infection there have been irregular attacks of fever lasting seven years and general disability associated with large patches of cutaneous oedema. But there has been neither mental symptoms nor tremor. He has been treated by atoxyl and tartar emetic

in the blood

**Prognosis**—This appears to be good *quoad vitam*

**Treatment**—The treatment is atoxyl administered as in sleeping sickness

**Prophylaxis**—Nothing can be said at the present time with regard to this

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## CHAPTER XLVI

# SOUTH AMERICAN TRYPANOSOMIASIS

Synonyms—Definition—History—Aetiology—Pathology—Symptomatology—  
Diagnosis—Treatment—Prophylaxis—References

Dr. *Chagas* (acute or  
*Chagas*,  
*megista*)  
He had fre-  
quently found a new trypanosome which he named *T. cruzi*, in the  
intestine of a species of *Lamus* which occurred in the State of  
Minas in Brazil. He also reported that he was able to infect

numbers in the houses of the poorer inhabitants. He noticed that  
the bite was painful, and that the insect was very voracious and  
also that it generally attacked people, especially children, at night,  
biting the face from which fact the inhabitants called it 'barbeiro'  
or 'barber.'

He suspected this *Lamus* of causing a disease marked by extreme  
anæmia which occurred especially among the children, and he  
was able to find in the blood of a two year old child during an  
attack of fever a trypanosome identical with *S. cruzi*, morpho-  
logically and biologically. Since then Chagas has not merely  
worked out the life history of the trypanosome in man and in  
the *Lamus* but he has also studied carefully the clinical and  
pathological aspects of the disease, while Vianna has reported  
upon the histopathology, Dias on the blood, and Guerreiro on the  
urine.

Chagas has also shown that in all probability the armadillo  
commonly called 'tatu,' and scientifically *Dasypus novemcinctus*,  
or less correctly *Tatusia novemcincta*, may be the reservoir for  
*Trypanosoma cruzi*, and that *Triatoma geniculata* (synonym, *Conor-*



*hinus geniculatus*) of the family Reduviidæ is one of the carriers of the same trypanosome. He also believes that *Triatoma infestans* and *T. sordida* may be carriers. It may be stated that *T. geniculata* lives in the burrows of the armadillo the flesh of which is rather a delicacy.

With regard to the history of the discovery of a trypanosome in man in South America Sambon informs us that in 1904 de Lacerda published a paper entitled *Etiologia de Beri Beri* in the *Brasil Medico* in which he

of  
pol

The reservoir of the trypanosome appears to be an armadillo—*Dasypus novemcinctus*.

In the blood three forms are seen the first with a large nucleus and loose chromatin and a terminal kinetonucleus the second narrower with an oval nucleus and dense chromatin the third with a long nucleus. The parasite undergoes schizogony in the lungs after which the merozoites enter the red blood cells and gony takes place in the bug found in the salivary glands of biting into the vertebrate and its life-history see

Chapter XIX p 427 )

the local inflammatory reaction appears and with the appearance of the trypanosomes in the blood the general symptoms make themselves evident. Whether the parasites produce any toxins or not is unknown but it is probable that they do so because of the

fatty degeneration described in the liver as well as because of

At present there is no evidence of any secondary infection being responsible for any of the essential pathological features of the disease

**Morbid Anatomy**—In an autopsy upon the body of a person dying from the acute phase of the infection a certain amount of serous effusion is remarked upon opening the abdomen. The liver is seen to be enlarged and to be in a state of fatty degeneration. The spleen is also enlarged hyperæmic and very soft as are the mesenteric glands. On opening the chest serous effusion is seen in both pleural cavities as well as in the pericardial sac. The pericardium shows signs of hæmorrhagic pericarditis while the enlarged heart is in a condition of intense myocarditis. The

between the leptomeninges and the cerebral cortex. The liquor cerebro spinalis is increased in amount. There is a generalized myxœdematous condition under the skin

cell and growing therein destroy the sarcoplasm and convert the body of the cell into a parasitic cyst without affecting the processes. When this cyst ruptures the now flagellate parasites

verted in  
inflamm  
seen to  
nervous system (vide Figs 642 and 643)

A similar invasion of the medulla or cortex of the suprarenal capsule and inflammatory reaction can also be seen in the kidneys, the hypophysis and the thyroid gland. In animals the parasites have been seen in the testicular tubules but they have not been noted in the human ovary

**Symptomatology** —There are two principal varieties of the disease —the acute and the chronic

In the *acute stage* the disease begins with a violent attack of fever in a young child or a new comer into the district. This fever shows

the lymphatic glands in various regions of the body but especially of the neck and fugitive œdemas in different parts of the body —as for example the forehead and extremities. The spleen enlarges and becomes painful, and the liver also becomes enlarged, and

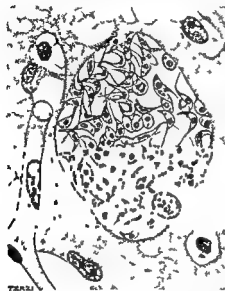


FIG 642—NEUROGLIA CELL OF BRAIN DISTENDED TO A CYST AND FILLED WITH *Trypanosoma cruzi* ( $\times 2000$ ) (After Vianna)



FIG 643—*Trypanosoma cruzi* IN A NEUROGLIA CELL OF THE BRAIN ( $\times 2000$ ) (After Vianna)

there may be signs of meningitis and also of albumen in the urine. After a time the attack of fever passes off only to return after periodical intervals. During an attack the typical trypanosomes can be found in the blood but during the apyrexial interval they are absent. After these attacks have lasted some time the child

- 1 The pseudo myxœdematous form
- 2 The myxœdematous form
- 3 The cardiac form
- 4 The nervous form
- 5 The chronic form with acute or subacute exacerbations

1 *The Pseudo Myxœdematous Form*—In this subvariety of the chronic stage there is usually hypertrophy of the lateral lobes of the thyroid gland more rarely a globular enlargement of the central lobe. This hypertrophy is usually well marked in quite young children but is by no means evident in older children. In young children the face is thin and the skin of a peculiar light bronze colour said to be quite different from the pallor of an anæmia. In older children the skin colour is violet bronze. These colourations are believed to be associated with a parasitic invasion of the suprarenal capsule.

There is enlargement of the lymphatic glands in the neck, axilla and groin, while the parotid gland is also often hypertrophied.

In young children the liver and spleen may be found to be enlarged but in older cases the abdominal signs are not well marked.

With regard to the circulatory system there may be tachycardia, sinus irregularities and an extra systole and the blood pressure may be lower. Convulsions have been noted and at times slight fever while the occurrence of conjunctivitis is also recorded.

2 *The Myxœdematous Form*—In this form the thyroid gland is atrophied and associated with the usual symptoms of myxœdema such as the rough skin, loss of hair, and the presence of a firm œdema not pitting on pressure together with an arrest of mental development in young children or a mental degeneration in older persons. The lymphatic glands of various regions are enlarged and there may be inflammatory eye affections.

3 *The Cardiac Form*—In the cardiac form there is disturbance of the heart action associated with arrhythmia, allarrhythmia, extra systole or sinus irregularities. The greater number of the cases would be classed under Mackenzie's *Rhythmus nodalis*.

4 *The Nervous Form*—Various brain and spinal cord symptoms are seen in this disease—e.g. spastic paralysis in the legs, athetosis in the arms, aphasia, pseudo-bulbar paralysis or suprabulbar paralysis. They are associated with the other symptoms of the disease.

5 *Acute or Subacute Exacerbations*—The principal feature of

trypanosomiasis. As a rule there is no globular anæmia but there is a definite diminution in the hæmoglobin and in the specific gravity. The leucocytosis is slight in the acute and exceptional in the chronic cases. In acute cases there is a macrolymphocytosis.

*Metabolism*—Guerreiro from careful experiments associated with urine analysis concludes that there is a true liver insufficiency in most forms of the disease but not in the cardiac form unless associated with other symptoms.

*Sequelæ*—Chagas considers that *infantilis* may be a sequel of the disease especially

↑

from which it can be recognized by the absence of the typical ova in the feces and the presence of *S. cruzi* in the blood though of course both infections may occur together.

It might also be mistaken for malaria during the febrile attack especially as there is splenic enlargement but the absence of the malarial parasite and the presence of *S. cruzi* in the peripheral blood will enable a diagnosis to be made.

In the chronic stage it may be mistaken for goitre especially when the myxœdematous or pseudo-myxœdematous symptoms are present, and the diagnosis will depend upon the discovery of the parasite or the history.

*Prognosis*—The prognosis is most serious in the acute attacks and the acute or subacute exacerbations. The severer cardiac forms are also of grave import.

*Treatment*—The indications for treatment are the same as those for African trypanosomiasis associated with treatment for hypothyroidism.

*Prophylaxis*—The prophylaxis must aim at the prevention of the *Larus* biting man.

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## CHAPTER XLVII

# THE KALA-AZARS AND PSEUDO KALA AZARS

General—Tropical kala azar—Mediterranean Kala azar—The pseudo-kala azars—Tropical febrile splenomegaly—Toxoplasmosis—Krempf's splenomegaly—Tropical afebrile splenomegaly—References

### GENERAL

THE present chapter is devoted to those fevers which are known

*Toxoplasma*

separately from Mediterranean kala azar and (this we shall do in the present chapter although the general tendency of modern

invite

XIX

to the

experimental work of Fantham and Porter (p 363) is having a direct bearing upon the unknown method of infection of man with the germs of kala azar

### TROPICAL KALA-AZAR

**Synonyms**—Indian Kala Azar Kala Jvar Kala Dukh Sirkari disease Sahib's disease Dum Dum fever Non malarial remittent fever Cachectic fever Tropical splenomegaly Tropical Leishmaniasis Internal Leishmaniasis

**Definition**—Tropical kala azar is a subacute or chronic febrile disorder characterized by splenic and often hepatic enlargement

occupied by the British a disease believed to be a very severe form of malarial cachexia was found to be endemic. This disease the Garos called 'kala jvar' which means the black fever so named



Leishmaniasis According to Christopherson the drug was discovered by Basil Valentine in the sixteenth century, and accidentally caused the death of several monks and thus acquired its name 'antimony'—*i.e.*, *anti-moine*, against the monk. In 191

method in the Sudan, and this has now become the recognized method of treatment

In addition to the above, much work has been done by the Indian

### China

**Ætiology.**—Kala azar is caused by a herpetomonad parasite called *Leishmania donovani* R Ross 1903 described on pp 369 370 which lives especially in the endothelial cells of bloodvessels and lymphatics, and is especially numerous in the spleen, the liver, and the bone marrow, but is also found in other organs such as the lungs and the kidney. Especially must be mentioned its presence in the mesenteric lymphatic glands, and in ulcers of the intestinal mucosa.

It can also be found in the blood of the patient during the acute stage of the disease.

blood cell thus looking as though it was contained therein. The parasites have been cultured from the blood by using the N.N.N. medium.

They are most abundant in the blood towards the fatal end of the illness and during fever or the presence of intestinal symptoms. They are said to have been found in the motions during an attack of kala azar dysentery, and also in the scrapings from intestinal ulcers. They have also been found in papules and ulcers in the skin.

As they occur in the peripheral blood and in the skin, it is possible that they may pass into the alimentary canal of some blood sucking arthropod, but these animals are often naturally infected with

point of view

As they occur in the peripheral blood and in the intestinal mucosa they can equally escape in the faeces in the form of cysts, and thus



get into water, from which they can be ingested by some aquatic arthropods, many of which naturally contain flagellates

The work of Laveran and Franchini, of Fantham and Porter, has demonstrated that these natural arthropodal parasites can by ingestion or by inoculation produce a fatal illness resembling kala azar in mammals. Archibald, experimenting with human kala azar parasites in the Sudan, has shown that monkeys can be similarly infected by feeding with kala-azar material, and this, together with the curious endemicity of the disease in the Sudan, and with Laveran, Franchini, Fantham, and Porter's researches, make the possibility of water carriage of cysts from infected arthropods to man worthy of consideration

“ *علاجها* ”

The predisposing causes appear to depend upon and be capable of explanation by, the habits of man. Thus the disease, when

people who are mainly affected are the poorer sections of the European and native communities. Season and sex appear to have *no influence*, but there is no doubt about the infection of the dwelling or perhaps its water supply, nor of the capability of the disease spreading from one dwelling to another, or from one water-supply to another.

**Pathology.**—Introduced into the body the parasite appears to enter the endothelial cells of a capillary bloodvessel or lymphatic, and to grow therein, and to increase in numbers by simple fission until a very large number—Leishman says upwards of 220—may be counted in one cell. The organs principally affected in this manner are the liver, spleen, bone-marrow, and lymphatic glands, and, to a less extent the pancreas, kidneys, suprarenals, testicles, and lungs.

The parasites may now escape from the enclosing cell by rupture, and are then taken up by the leucocytes particularly by the polymorphonuclears, but also by the mononuclears and rarely by the eosinophiles by means of which they appear in the peripheral blood even in early cases, but are much more common late in the disease, especially if there is diarrhoea due to ulceration of the intestine, in which condition the polymorphonuclear leucocytes are increased in numbers in the peripheral blood, and many of them contain parasites. The further development has still to be worked out, as all that is definitely known is that in cultures the

bone-marrow as well as the ulceration of the skin and intestinal mucosa because sometimes and in an inconstant manner it can

be the cause of the refractory nature of certain animals to the disease and the limitation of infection in endemic communities. After the infection has obtained its hold on the body as a rule the struggle for immunity becomes less and less and disappears eventually.

By some means or other the parasite irritates the organ it infects causing marked changes in the spleen, liver and bone-marrow and also causing ulceration of the intestine and skin.

**The Blood**—The examination of the blood is most important because firstly the parasite may be found in a leucocyte if carefully looked for even in the early stages of the disease secondly the leucocytic changes are of the utmost importance. There is marked anaemia—54.2 per cent of Rogers' cases giving from 4,000,000 to 2,500,000 corpuscles per cubic millimetre—and the haemoglobin is reduced in proportion to the erythrocytes the colour index being normal. There is a most marked leucopenia and Rogers reports that in 47.1 per cent of his cases the leucocytes were 1,000 or less, in 30.3 per cent 1,000 to 2,000 and in 22.6 per cent 2,000 to 3,000. The proportion of white to red according to the same author is less than 1:1,500 in 67.9 per cent or if inflammatory cases are excluded in nearly 90 per cent of the cases he examined. There



FIG. 644.—INDIAN KALA AZAR.

The distension of the abdomen by the greatly enlarged spleen should be noted. (Photograph of a case in the Tropical Clinic, Colombo.)

is a reduction in the polymorphonuclear leucocytes and in the eosinophiles and an increase in the mononuclear leucocytes and lymphocytes. The diminution of the polymorphonuclear leucocytes is thought to explain the tendency to bacterial infections. The

diminished—a fact which may be of some secondary diagnostic importance.

*The Urine*—The urine in our cases did not show anything abnormal.

*Morbid Anatomy*—The body is much emaciated and there is marked muscular atrophy together with œdema, enlargement of the spleen and often of the liver, ulceration of the skin and intestine, sometimes hæmorrhage in various places and generally the presence of some complication. The spleen is greatly enlarged, firm and deep red in colour though it may at times show malarial pigment.



FIG 645—TEMPERATURE CHART OF KALA AZAR FROM A SUDAN CASE  
(After Christopherson)

+ Leishman bodies found    XXXX injection of tartar emetic

The capsule and septa are thickened and the whole organ is congested with blood and contains numerous mononuclear cells and macrophages full of parasites.

In the liver which may or may not be enlarged the most marked changes are in the intralobular capillaries which are dilated and contain macrophages derived from their endothelial wall full of parasites while the liver cells are atrophied and degenerated.

shows the cicatrices of old ulcers.

*Symptomatology—Incubation*—The incubation period appears to be very variable and indeed it is difficult in a chronic disease of this nature to decide when it first begins. It is said to range from ten days to three weeks or several months.

*Onset*—The onset may be heralded by a rigor which may be

slow or quick

The attack however may begin with a continuous fever which shows two fluctuations in the twenty four hours which Rogers considers as almost diagnostic of the disease. In other cases the disease may begin with gastro intestinal disturbances or with dysenteric symptoms or quietly without any marked initial stage the patient gradually developing an enlarged liver and spleen anæmia and weakness

*Course*—The course of the disease after the decline of the tem

perature

On examining the swollen abdomen the enlarged spleen may be felt reaching almost to the pelvis while the enlargement of the liver may be marked or may be absent. In this condition intestinal disturbances in the form of diarrhœa or dysenteric attacks are common and may be due to the actual disease or to complication with true dysentery. Dyspeptic symptoms may also be present.

Hæmorrhages may occur from the nose the gums the stomach the bowels or under the skin. Papular eruptions are to be seen especially on the thighs and ulcers may be present. The weakened emaciated patient may now die of asthenia but more usually the

vasion by pathogenic bacteria for septic infections such as cancrum oris or lung infections—for example pneumonia phthisis and pleurisy—or abdominal troubles of the nature of diarrhoea dysentery and cystitis are not uncommonly met with and may cause the death of the patient. Sometimes after a severe attack of septicaemia or some other complication the disease is found to be cured but this is rare.

**Diagnosis**—The only certain method of diagnosis is to find the parasite and as Donovan and Patton have reported its frequent occurrence even in early stages in the peripheral blood this should be possible especially if aided by dilution with normal saline solution and centrifugulization and examination of the leucocytes. In our experience the search for the parasite in the leucocytes of the peripheral blood requires an extremely long time and is often negative. If the parasites cannot be found in the blood an attempt may be made to find them by the examination of the exudate obtained by exciting artificial pustulation of the skin by some irritant as suggested by Cummins. Failing this there is puncture of the spleen or of the liver and withdrawal of blood which can be examined by the microscope. The diagnostic puncture of the spleen in the tropics is however not to be undertaken lightly because splenic enlargement due to leukaemia is by no means unknown and puncture of the spleen in this disease or indeed in that of chronic malaria may lead to most unfortunate results. The blood of the peripheral circulation should therefore be examined to exclude leukaemia.

Certainly the first thing to do is to examine the peripheral blood and exclude leukaemia. Secondly the coagulability of the blood should be tested by Wright's method and if found to be decreased the puncture should not be performed. Thirdly if the puncture is to be carried out the liver should be chosen for exploration not the spleen particularly in the later stages in which haemorrhages are to be feared. In the early stages there may not be so much risk but it must be done with the greatest care aseptically and the patient must be kept at rest for some time afterwards the site of puncture being covered with an aseptic pad and a firm bandage. The syringe should be sterile and perfectly dry. Rogers recommends that a dose of 30 grains of calcium chloride in a couple of ounces of water be administered directly after a puncture in order to promote coagulability of the blood. Attempts at cultivation from the blood and inoculations into susceptible animals may also help rats and monkeys being used by preference.

daily double remission of the fever (2) absence of constitutional

enlargement of the spleen (5) great leucopenia especially in relation to the erythrocytes which however may also be found in typhoid and malaria, (6) increase in mononuclear leucocytes (7) presence of *Leishmania donovani* in the leucocytes

In advanced cases the diagnosis has to be made from malarial cachexia and ankylostomiasis by (1) the presence of *Leishmania donovani* in the leucocytes of the peripheral blood or in the juice from the liver and spleen (2) by the absence of the typical febrile attacks of subtertian or tertian fever (3) by the absence during the febrile attack of malarial parasites (4) by the absence of ancylostomes or if they are present by the continuation of the symptoms after their expulsion Mixed infections of kala azar and malaria may occur

**Prognosis**—The prognosis is much less serious than before the introduction of the tartar emetic treatment Formerly the mortality was about 98 per cent It is true that some people recover after having nearly died from a complication or more rarely without this episode but why they recover is not known

Leucocytosis and increase of the polymorphonuclears are considered to be good signs while leucopenia and polymorphonuclear decrease are bad signs Complications of course increase as a rule the gravity of the prognosis

**Treatment—Essential Treatment**—As soon as a diagnosis is made give *tartar emetic* either—

- (a) Intravenously (this is the method to be preferred)
- (b) Intramuscularly
- (c) Orally combined with (a) or (b)

so

an

method of treatment

*Dose for Children*.—This is as follows —

INTRAVENOUS DOSAGE OF 1 PER CENT TARTAR EMETIC

Age	Dose	Number
Under one year	$\frac{1}{2}$ c c	One daily for seven days D tto D tto D tto
One to five years	1 3 c c	
Five to ten years	1 5 c c	
Ten to sixteen years	1 $\frac{1}{2}$ c c	

**Important**—The sterilization of the tartar emetic solution must be made in flowing steam on two or three consecutive days and must not be performed in an autoclave in which the drug is liable to decomposition and may then cause serious symptoms. Some authorities advise using a solution merely filtered through a Chamberland filter One of us has used a solution containing  $\frac{1}{2}$  per

cent carbolic, which in practice renders unnecessary a sterilization by heating

*Intramuscularly*—Intramuscular injections are painful and often become inflamed. The following solution may be used—

Tartrate of antimony	8 grains
Carbolic acid	10 minims
Glycerine	3 drachms
Bicarbonate of sodium	$\frac{1}{2}$ grain
Distilled water	1 ounce

The dose is  $\frac{1}{2}$ –1 cubic centimetre every other day injected intramuscularly into the gluteal region

Martindale's formula may also be used—

Antimonii oxidi	gr $\frac{1}{5}$
Glycerin }	
Aq. dest. }	aa ℥xv

One ampoule

*Combined*—Oral administration may be combined with intravenous or intramuscular injections. The following mixture may be given—

Tartrate of antimony	5 grains
Bicarbonate of sodium	30 grains
Glycerine	1 ounce
Chloroform water	1 ounce
Water	to 3 ounces

The dose is one to two teaspoonfuls in water three times a day

Rogers regards sodium antimonyl tartrate given intravenously as being more efficacious than tartar emetic. Colloidal antimonial preparations have been recommended.

**Symptomatic Treatment**—*Hæmorrhagic symptoms* may be treated by calcium lactate in 10 grain doses twice or three times a day. *Diarrhœa* may be combated by bismuth subnitrate in 10–12 grain doses with or without 5–10 grains of salol every four to six hours as may be required. *Intestinal parasites* should be looked for and treated as prescribed in the chapters pertaining to the different forms. *The heart* must be watched and cardiac tonics or saline injections given if required (*vide* Treatment of Malaria p. 1188).

**General Treatment**—The patient should be kept in bed and well nursed during this treatment.

**Diet**—The diet should be good and nourishing, but if there is much diarrhœa it is necessary to restrict it to milk, Benger's food and the like soups etc.

**Prophylaxis**—As the method of infection is unknown, all that can be done is firstly to segregate the sick and carefully disinfect his motions, as well as protect him against blood-sucking arthropods. Secondly, to remove the healthy from the infected area and to disinfect or destroy the clothing, furniture and houses while a complete change of the drinking water supply is essential. If this latter cannot be done and the water supply is a well, it may be

## MEDITERRANEAN KALA-AZAR

**Synonyms**—Infantile kala azar, Infantile leishmaniasis Mediterranean leishmaniasis Febrile splenic anæmia (Fede) *Anæmia infantum* a *Leishmania* (Fianese) *Leishmania anæmia* (Jemma and di Cristina) *Marda tal biccia* (Malta) *Ponos* (Greece) *Malattia da mensa* (Sicily)

**Definition**—Mediterranean kala azar is a subacute or chronic specific disease due to *Leishmania infantum* Nicolle and clinically closely resembling tropical kala azar but occurring in temperate or subtropical climates

**Historical**—Fede several years ago described in Italy a form of splenic anæmia among young children characterized by irregular

symptoms of Fede's splenic anæmia with kala azar and described parasitic bodies in the spleen of the affected children morphologically identical with *Leishmania donovani*. Later Nicolle suggested for the disease the name of infantile kala azar and completed the study of the parasite which he called *Leishmania infantum*. Gabbi considers the disease to be identical with tropical kala azar

the northern regions of Africa and perhaps Egypt. Future investigations will probably show that it is endemic in many countries.

**Ætiology**—The malady is due to *Leishmania infantum* Nicolle. The description of this parasite will be found on p. 373. Nicolle has succeeded in reproducing the disease in monkeys and less typically in dogs. He has also found that dogs may be spon-

ted experimentally by *Leishmania* but to day doubt is cast upon canine leishmaniasis being the same disease as that in man and the flea infection of man is also considered to be doubtful.

The majority of the cases occur in young children of two to three years of age among whom there is a slight preponderance of males. The disease sometimes occurs in more than one member of a family and more of



the spleen and hypertrophy of the islands of Langerhans in the pancreas. In the bone marrow there is hyperproduction of the

turbance of the alimentary canal such as an attack of vomiting and diarrhoea when the spleen may or may not be found to be

rigors

The child becomes pale, ceases to be interested in its games and suffers from attacks of diarrhoea alternating with periods of constipation, from attacks of irregular fever separated by apyretic intervals and from epistaxis.



FIG. 646—*Leishmania infantum* NICOLLE  
(After Marznowsky)



FIG. 647—CHILD SUFFERING FROM INFANTILE KALA AZAR  
(After Marznowsky)

*Course*—After the above symptoms have lasted some time the spleen begins to enlarge and presently protrudes from under cover of the ribs; the attacks of fever become more marked, hæmorrhages from the nose and gums and into the skin are seen, and the diarrhoeic or dysenteric attacks become pronounced. The child now wastes

subnormal temperatures

The alimentary canal is always disturbed, but the appetite is preserved and may even be increased, although the little patient

may occur and the last may appear on the face or on the genitalia. Noma appears to be not uncommon in Greece.

ments and may be altered in position from side to side and up and down by manipulation. There is not however any constant relationship between the progress of the disease and the size of the spleen. Jemma, Di Cristina and Critien state that it diminishes with a persistent and profuse diarrhoea especially during the last few days of life.

There is not always a concord between the temperature and the pulse rate on the contrary the latter is almost constantly rapid even during the apyrexial intervals but may rise to 150 to 160 beats per minute during attacks of fever. Hæmic murmurs may occur over the heart but are rare. The blood is pale and shows a decrease in the number of erythrocytes (1 500 000 to 3 000 000) and in the hæmoglobin (below 50 per cent) which however is reduced in proportion to the red corpuscles and also in the leucocytes

nuclear cells which make up the remaining 20 to 30 per cent. There is usually some poikilocytosis and anisocytosis but nucleated cells are rare or absent. The opsonic index is diminished below that which is normal for a healthy child and is especially low for *Bacillus coli communis* which is held to be responsible for the

are influenced by the position of the patient and may occur at any stage of the disease. At times they are painful and may show signs

and is not tender.

As the spleen and liver enlarge the abdomen also enlarges and becomes prominent while the superficial veins may stand out distinctly and there may be a slight degree of ascites at times.

The urine is usually normal but there may be slight signs of albu



undulating type the enlargement of the spleen is much greater the articular symptoms are lacking and Wright's agglutination test is negative

*Enteric Fever* —From enteric infantile kala azar differs by the splenomegaly by the irregular fever by the absence of Widal's reaction

*Malaria* —The absence of the typical blood parasites and the fact that quinine has no influence on the irregular fever are points of diagnostic value

*Other Forms of Splenic Anæmia* —From the splenomedullary

splenomegaly found in rickety children by the absence of deformity of the bones and by microscopical examination

*Prognosis* —The prognosis is much more favourable since the introduction of the tartar emetic treatment the mortality having been reduced from 90 per cent to less than 20 per cent

*Treatment* —This is the same as for tropical kala azar (p 1297)

*Prophylaxis* —As canine leishmaniasis (p 377) is now considered to be a separate disease and as the flea is doubtful as an infective

of infection being correct to make the simple methods of prophylaxis of such a fatal disease imperative even before the full proof of the researches has been obtained

### THE PSEUDO-KALA-AZARS

These are febrile and afebrile diseases which resemble kala azar in that they are associated with splenomegaly anæmia and often emaciation They can be divided into —

- Tropical febrile splenomegaly
- Toxoplasmosis
- Krempf's splenomegaly
- Tropical afebrile splenomegaly

### TROPICAL FEBRILE SPLENOMEGALY

*Synonyms* —Tropical splenomegaly Pseudo kala-azar Esplenomegalia tropical (Columbia) Wenku (Karonga) Gobora or Tebu (New Guinea)

*Definition* —A chronic irregular febrile disorder of unknown

that infection cannot be found either during life or after death. Though well known, there is but little literature upon the subject. Woolley first gave an excellent account of the disease as seen in

as that described by Breinl, in 1915, in New Guinea. In 1916

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canal

secondary, but we would r  
forms of polyfibrosis, and  
order was in the spleen ar  
signs were secondary

**Morbid Anatomy.**—On post-mortem examination, the body is seen to be emaciated as a rule, but there may be œdema of the feet and legs, and there may be ascites if the liver is seriously affected. Upon opening the abdomen, the principal object of interest is the extremely enlarged firm spleen. The liver may be enlarged and smooth, a cirrhosis, may be splenic, or ulcerative.

*Hæmorrhages may at times be found in different parts of the body.* Microscopical examination reveals hyperplasia of the lymphoid elements of the spleen, associated with hyperplasia of the fibrous tissue, dilatation of the vascular sinuses, and sometimes hæmorrhages. The microscopic examination of the liver shows the usual

dysentery, or of attacks of fever may be obtained. Usually the patient comes to the hospital complaining of weakness and vague

treme, the average number of red corpuscles being between 2,500,000 and 3,500,000, while microcytes, megalocytes, and polychromatophilia are not unusual. The leucocytes are more or less normal.

Sometimes accompanied by jaundice. Death in the first stage is, in our experience, not rare, and may be due to hæmorrhages or exhaustion.

#### febrile splenomegaly

**Diagnosis.**—The leading features of the disease are the great enlargement of the spleen, associated with wasting and irregular fever, in people in whom examination fails to reveal any obvious parasitic cause. Tropical febrile splenomegaly must be differentiated from kala azar by the absence of *Leishmania donovani* in the spleen pulp as obtained by puncture. It can also be distinguished from chronic malaria by the absence of the typical parasites

from cirrhosis of the liver. From leukæmia it is easily distinguished by the absence of Leishmania in the splenic juice. The disease can be distinguished from Banti's disease by the febrile attacks, and apparently splenic removal does not effect a cure.

**Prognosis.**—The illness is very chronic, but the prognosis is bad, as no cure is at present possible, and the patient tends to go from bad to worse.

**Treatment.**—Arsenical injections are the most valuable, and salvarsan may be tried. Removal from the endemic area is advisable.

**Prophylaxis.**—As the ætiology is unknown, nothing can be said under this heading.

#### TOXOPLASMOSIS

In 1913 Castellani recorded a case of splenomegaly associated

**Morbid Anatomy.**—The body was much emaciated, and the principal feature was the greatly enlarged, smooth, not very hard

spleen, which was reddish in colour. No malarial parasites could be found, but there were some light yellowish pigment granules, quite different from malarial pigment.

**Toxoplasma Pyrogenes.**—This was rarely found in the blood but

de  
103° to 105° F at times. The attacks of fever do not start with shivering and the fall is not associated with sweating. The spleen is much enlarged and hard, while the liver is also enlarged but neither organ is tender on pressure. All the other organs are normal, and there is no enlargement of the lymphatic glands.

**Blood Counts**—The red blood corpuscles in an advanced case number 2,000,000 the leucocytes 5,200 per c mm. A few nucleated red cells are present and basophilia and chromatophilia are marked. The leucocytic count is as follows—Polymorphonuclear leucocytes 50 per cent, lymphoc 7 per cent, eosinophiles, 3.

No malarial parasites for typhoid, the paratyphoids and Malta fever, were absent.

The urine sometimes contained a trace of albumen.

**Course**—The case grew gradually worse emaciation set in and the patient died.

**Treatment**—Quinine was given by the mouth and intramuscularly in doses of 30 to 60 grains a day without effect.

### KREMPF'S SPLENOMEGALY

In 1917 Krempf described a case of splenomegaly in a young Chinaman. He suffered from a malarial infection and stated that in his village near Tientsin splenomegaly was frequently observed in both sexes and at all ages.

On making a splenic puncture Krempf found bodies either enclosed in red cells or free in the plasma. They were only found in the spleen.

The red cells were deformed and contained a capsule 10 × 5 microns in size inside which lay a vermicular sporont often curved like the letter U. Extracted from a red corpuscle these bodies measured 20 × 15 microns.

These bodies were believed to be the sporonts of a haemogregarine, and were named *Haemogregarina hominis* Krempf 1917. No further history of the case is given.

Recently Raubaud examining the blood of a lady who had resided for two years in the Congo observed that some red cells contained a haemogregarine 9.11 μ by 2.835 μ which differed from Krempf's parasite by having a crescenting not vermicular shape. Raubaud has named it *Haemogregarina inexpectata*. There was no fever and no enlargement of the liver or spleen. The blood however showed a marked mononucleosis.

### AFEBRILE SPLENOMEGALY

**Synonym**—Pseudo Banti's disease

**Definition**—A chronic afebrile disorder characterized by splenomegaly and severe anaemia.

History — There is no literature on the subject as far as we know  
 as to Ceylon and India

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#### Léger's Disease

Léger has recorded a case from Guiana of prolonged fever with great enlargement of the liver in which he found organisms of two types. Some Others

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complication than a feature of the disease. The liver is enlarged and tender, and so is the spleen, which may reach a considerable size. The heart sounds are normal, but the pulse rate is quick,

bronchitic expectoration, and râles, which can be heard over the chest and trachea. The respirations follow the pulse-rate, being increased to 48 to 50 per minute if the temperature is high. The pains in the muscles and joints continue, and sleeplessness may be uncommon.

About the middle of the attack a rigor intervenes, with violent perspiration, or diarrhœa, with or without epistaxis, and with a sudden rapid fall of temperature, while the pulse and respirations also return to normal and the patient falls

ment of the illness, the relapse occurs, beginning with a rigor

The complications are numerous, affecting the lungs, pneumonia, or the alimentary tract, hæmatemesis, while cerebral hæmorrhage, conjunctival hæmorrhage, iritis and corneal ulcers have all been recorded.

Abortion often complicates the first relapse in pregnant women.

**Diagnosis.**—In the first instance, before the relapsing character has appeared, the disease requires to be diagnosed from malaria, typhus, typhoid fever, yellow fever, and seven days' fever. The principal positive signs indicating relapsing fever are —(1) Presence of the spirochaetes in the blood, (2) agglutination or Lowenthal's

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Differ. diagnosis is made by the absence of spirochetes, by the absence of the characteristic rash, and by the fever having started gradually.

by the fever having started gradually. *Typhus* can be separated by the absence of spirochetes, by a negative Lowenthal's reaction, and by the presence of its characteristic rash. *Yellow fever* can be diagnosed by its black vomit though at first the differentiation may be impossible without a microscopical examination of the blood. *Dengue fever* is characterized by its slow pulse and, of course by the absence of spirochetes. *Weil's disease* may be distinguished by the more marked jaundice and by the different nature of the spirochetes, which are seldom found by the simple microscopical examination of the blood.

**Prognosis.**—The prognosis is usually favourable. Marked jaundice is a bad sign, while pregnant women generally abort. The mortality appears to vary considerably, being, according to Murchison only 4 per cent in the United Kingdom while, according to Sandwith, it is 14·4 per cent in Egypt, which is nearly the same percentage as that reached in Russia. The causes of death are toxæmia in the first attack, and collapse in the first intermission, but it may be caused by one of the above mentioned complications.

**Treatment.**—This may be discussed under the following headings—

- 1 Specific Treatment
- 2 Symptomatic Treatment

**Specific Treatment**—Salvarsan or neosalvarsan or their substitutes may be administered either by intramuscular injection or, better, intravenously. This is a specific treatment, most efficacious, but care should be taken not to inject a large dose, as certain patients, especially it seems those suffering from Asiatic relapsing fever stand the drug badly, cases of death having been recorded even after a medium dose such as  $7\frac{1}{2}$  grains (0·5 gramme). According to Mouzels, an intravenous injection of 4 or 5 grains (0·3 gramme) does not give rise to any unpleasant symptom, and is generally sufficient to make the spirochetes disappear from the blood and cure the attack. If, however, another attack of fever develops a second injection of the same dose may be given.

**Symptomatic Treatment**—Pains in the head and muscles may be relieved by small doses (2 to 3 grains) of salicylates, aspirin, antipyrin, or by quinine. If these pains are very severe opium or a hypodermic injection of morphia may be necessary. *Epigastric pain* may be relieved by fomentations sprinkled with tincture of opium, while *vomiting* should be treated with ice, champagne, and bismuth mixtures, though occasionally morphia or codeine may be required. Effervescent ammonium carbonate mixtures are often grateful.

A dry, troublesome cough may be relieved by codeine or small

doses of heroin, in other cases an expectorant mixture will be found useful. Constipation must be treated by laxatives or enemata and high temperatures by cool sponging, which, however, will but seldom be required. The complications must be met by the treat-

of typhus (p 1338)

### THE RELAPSING FEVER OF NORTH AFRICA.

**Synonyms.**—Algerian relapsing fever Egyptian relapsing fever, Arabic Homa el Hugga Homa en Naxy Naushah

**Definition.**—An acute specific fever caused by *Spiroschaudinnia berbera* Sergent and Foley, 1910, and spread by the agency of

Balfour, and Graham Smith, have published excellent accounts of the disease. In 1910 Sergent and Foley differentiated *S. berbera* Sergent and Foley in a case in South Oran. The transmission of the disease by lice has been worked out by Nicolle, Blaizot, and Conseil. Toyoda's immunological experiments show that this

Tunis, Tripoli,

spread by the agency of lice, as shown by Nicolle, Blaizot, and Conseil (see p 447)

**Symptomatology.**—The length of the incubation period is not known, but is believed to be more than twelve days. The fever which may be associated with rigors reaches its height during the first twenty four hours, and afterwards shows morning remissions. The spleen enlarges, and the liver becomes tender and painful in some cases, but jaundice is generally absent. Vomiting is present, but diarrhoea is absent. The attack is apparently not very severe. Apyrexia lasts from two to nine days and is followed by one two, or, more rarely, three relapses.

**Diagnosis.**—The spirochaetes must be found in the blood as the cases may occasionally resemble cerebro-spinal meningitis and acute rheumatism.

**Prognosis.**—This is usually good, the mortality being nil in fifty cases.

**Treatment.**—This is the same as for the other relapsing fevers.

## THE RELAPSING FEVERS OF ASIA.

There are probably a number of relapsing fevers in Asia, but we only know two which have lice as carriers, and these are —

- 1 The Indian relapsing fever.
- 2 The Manchurian relapsing fever.

## The Relapsing Fever of India.

**Definition.**—An acute specific relapsing fever caused by *Spiroschulinnia carteri* Manson, 1907, and spread by the louse in all probability

be included under this term, and recent research shows that several varieties of relapsing fever probably exist in India. Types of relapsing fever, which may possibly be different from the Indian ones, occur in China and French Indo China, while the Arabian type may be identical with the West African relapsing fever.

**Climatology.**—The real home of the disease appears to be the

quarter years. It can also be inoculated into monkeys, but not into rats and mice. Bugs are capable of retaining it alive in their

— ckie has brought  
being a carrier.

*S. carteri* is separated from *S. duttoni* by the latter being far more easily inoculable into animals and producing numerous relapses

Strong, experimenting with white mice, has come to the conclusion that the Indian *Spiroschaudinnia* is closely allied to the European and North American types

**Pathology.**—The morbid anatomy resembles that of the European type

**Symptomatology.**—In accidental inoculations in the post mortem room the incubation period varies from three and a half to seven days, during which prodromata resembling those of the European type may occur

The onset is generally sudden, but in most cases without the rigors defined in Europe, though chilliness occurs and the disease progresses as in the Obermeyer fever

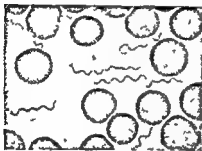


FIG 649—SPIROSCHAUDINNIA FROM A CASE OF ASIATIC RELAPSING FEVER

(From a microphotograph by J J Bell)

to be seen in the blood, but Carter and Pisani have described peculiar structures The first relapse occurs about the fourteenth day of the disease, and the seventh of the intermission and resembles the first attack but the temperature may reach a higher level and the illness is shorter, ending in a crisis

The second intermission may last about ten days, being longer than the first, and a second relapse takes place often commencing with chills The liver does not markedly enlarge, but the spleen increases in size The fever is remittent or intermittent, and the crisis is not marked It is now rare for the disease not to end, but after take 1  
four d  
ends in a fourth relapse, lasting only two days

to subnormal however, on the sixth or seventh day, which is associated with profuse perspiration and polyuria, instead of the patient feeling better, he often becomes collapsed, with a small weak pulse and a cold clammy skin in which condition he may resemble at first sight a cholera patient In the first intermission which lasts from three to twelve days—generally about eight days—the patient improves slowly, there being much debility and in one case in six there is a sudden temporary rise of temperature after the crisis Spirochaetes are not

use may  
one to  
n days

With regard to the frequency of the relapses Rogers gives the following percentages —

Without relapse	23.8 per cent
With one relapse	49.2
With two relapses	20.0
With three relapses	5.0
With four relapses	2.0

**Varieties**—The typical course may be varied in about 25 per cent of cases and present (a) a *short irregular remittent fever*, c fever malaria

o called  
and to

some extent yellow fever showing deep jaundice with an eruption of red spots. The temperature is irregular and the pyrexia prolonged prostration comes on early and may develop into a status typhosus. The death rate of these cases is high being 70 per

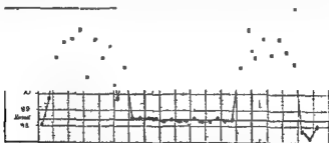


FIG. 650.—TEMPERATURE CHART OF THE RELAPSING FEVER OF INDIA (Chart made by Major Archibald)

at The ... ..

the blood

Diagnosis—A prolonged ... ..

relapse and the remainder from complications.

**Treatment**—The treatment and diet differ in no respect from that already laid down except that these patients often stand large doses of silvarsan badly and therefore one should not give more than 5 grams of the drug by intravenous injection, more over the disease being of a severer type than the European form,

and sudden heart failure being common, cardiac stimulants should be given during the attack, and preparation made for preventing the collapse by means of hypodermics of strychnine and ether, or camphor in ether, as well as by hot bottles, blankets, etc

**Prophylaxis.**—See remarks with reference to the European type

#### Manchurian Relapsing Fever.

This type of relapsing fever is due to a spirochæte which Toyoda in 1916 demonstrated by immunity experiments to be distinct from the African and the European types. The organism is short 7.20 microns by 0.4 micron and its spirals number 4.8. It is spread by lice.

Liver enlargement and albuminuria are physical signs of importance. The first paroxysm lasts five to thirteen days, the first interval two to fourteen days, the second attack one to nine days, interval two to thirteen days, the third paroxysm lasts one to six days, the third interval two to ten days, the fourth paroxysm lasts two to six days.

The mortality rate is 5.3 per cent. Two paroxysms are common.

## THE TICK GROUP

### THE RELAPSING FEVERS OF AFRICA.

The tick relapsing fevers of Africa may be classified as follows—

- 1 West African relapsing fever
- 2 East African relapsing fever

#### 1 The Relapsing Fever of West Africa

**Synonyms**—Tick fever (Livingstone) African tick fever

**Definition**—An acute specific relapsing fever caused by *Spirochaudinnia duttoni* Novy and Knapp 1906 and spread by *Ornithodoros moubata* Murray

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ap  
m

nature was spoken of as occurring in Africa. Nabarro in August 1903 was the first to observe a spirochæte in human beings in Uganda, but as his publication through no fault of his own did not appear till much later, his discovery was forestalled by those of Ross and Milne in 1904 and Dutton and Todd also in 1904 who found the cause of the tick fever to be a spirochæte, the latter observers also proving that it was introduced into the blood by the bite of a tick—*Ornithodoros moubata*. Since that date Koch, Todd, Novy and Knapp, Breml and Kinghorn, and others, have studied the disease carefully. Frankel has proved by biological tests that the spirochæte of East African relapsing fever differs from *S. duttoni*, as observed in West Africa, and Nuttall proposed

the name *S. rossii* Nuttall 1908 for this new species but recent

others have described a spirochaetiasis in Colombia which is caused by a spirochæte morphologically resembling *S. duttoni* and said to be spread by *Ornithodoros turicata* with reference to which it may be noted that Buldo v observed relapsing fever in the Andes in 1865 Robledo asserts that it is spread by the bite of *Ornithodoros megnini*

fever or allied to this West African form as it may be spread by an *Ornithodoros*

**Ætiology**—The cause of the fever is *S. duttoni* Novy and Knapp 1906 which is proved to be distinct from the other spirochætes because an animal immunized against one of these is capable of being successfully inoculated by it *S. duttoni* can be inoculated into a number of animals—namely dogs goats sheep rabbits guinea pigs rats and mice—but not into cats chickens pigeons or goldfish The spirochætes can pass through the placenta into the circulation of the foetus which they infect These organisms are easily seen in the blood during an attack but disappear during an apyrexial interval The spirochæte is described on p 444

The life history in the vertebrate has been worked out by Breinl who as already described showed that just before the crisis the spirochætes become encysted and undergo schizogony into small bodies from which the new generation develop Leishman has demonstrated that when the spirochætes pass into the intestinal sac of the tick they lose their motility and their characteristic appearance while the central core of chromatin segments into small masses which are set free into the lumen of the gut These small bodies which resemble small rods or are rounded like micrococci appear to multiply in the body of the tick and to pass into the cells of the Malpighian tubules and into the tissue of the ovary In the latter position they enter the immature eggs and can be traced through all stages of development into the adult

chromatin bodies that the disease is carried from the egg to the new generation of ticks and that infection of man does not take place via the salivary glands but by the small bodies gaining access



to the wound produced by the tick's bite by being voided in the Malpighian secretion passed by the tick during feeding or perhaps by regurgitation of the intestinal contents. More recently Leishman and Hindle have shown that the tick produces infection only as the result of its infected faeces contaminating the tick bite. This

hence predispose to the disease

**Pathology**—Very little can at present be said as to the pathology. The post mortem reveals only an enlarged firm spleen while smears taken from it

**Symptoms**—  
investigation  
good work has been done on the subject by Dutton Todd Ross and others

**Incubation**—The period of incubation is usually about seven days but it may extend to eleven or twelve days

The tick bite may be accompanied by local inflammatory symptoms but in some cases the bite is not even noticed. According to Wellman natives believe that when the tick bite is accompanied by a severe local reaction the individual probably escapes fever and Nuttall calls attention to the possible protective effect of a local reaction

Usually mental heaviness lack of activity profuse sweating and

and a slight rise of temperature. In a few hours the temperature will have risen to 103° to 105° F. associated with headache pains in the back and limbs and intense pain in the region of the spleen and chilliness. There is vomiting first of food and then of bile with often diarrhoea and even at times streaks of blood in the motions. The spleen is generally found to be enlarged and spirochaetes in scanty numbers occur in the peripheral blood but may be hard to find

**Course**—The next d

an evening rise during which the pains increase and the patient may become delirious. The liver does not enlarge but the spleen projects below the costal re is a slight decrease

red cells and a very marked

symptoms last three to four days and end by a crisis marked by profuse sweats and a fall of temperature below normal. On the day before the crisis there is a pseudo crisis with a fall of temperature but no improvement in the symptoms.

**Intermission**—The patient feels weak and tired but slowly regains his appetite and strength and no parasites are found in the blood. The disease may now terminate or the intermission may last from one to twenty one days according to Ross but five to eight days is more usual.

**Relapse**—The first relapse begins with a rise of temperature and a return of the original symptoms and also of the parasites into the peripheral blood. After lasting three to four days it ends in a crisis marked by sweating and showing a pseudo crisis on the preceding day.

Intermissions and relapses follow one another regularly or irregularly the intervals being from one day to two months and the relapses usually lasting three days and showing a pseudo crisis on the second day and a crisis on the third day. As many as five to eleven relapses may take place and end by reducing the patient both in weight and strength. Edema of the eyelids has been noted in the relapses.

**Complications and Sequelæ**—Iritis is frequently observed.

**Clinical Varieties**—According to Ross there appears to be a marked difference in the severity of the attack in new comers such as Europeans and natives. Though the attack shows much the same symptoms in natives as in Europeans it is often far less severe and the spleen may not enlarge. The attack frequently lasts twenty four to forty eight hours and ends by crisis after which the patient rapidly recovers as a rule without a relapse.

**Death**

**Diagnosis**—This can only be made by finding the spirochetes. A wet film method of agglutination can be applied in doubtful cases.

**Prognosis**—This is usually good for both the natives and Europeans but death may occur in both races and is signalled by a rapid fall of temperature without improvement of the symptoms.

No figures have so far been recorded with regard to the mor-

tal

ad

of

**Prophylaxis**—The prophylaxis is based on the avoidance of localities where ticks abound and the destruction of these parasites. Koch rightly advised that Europeans should camp at least 20 to 30 yards away from infected native huts and rest houses.

### ■ The Relapsing Fever of East Africa.

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### THE TICK FEVER OF MIANA, PERSIA.

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## THE RELAPSING FEVERS OF AMERICA.

The relapsing fevers of America comprise —

- 1 North American relapsing fever
- 2 Central American relapsing fever
- 3 South American relapsing fever

## 1 Relapsing Fever of North America.

**Definition.**—An acute specific fever caused by *Spiroschaulinia novy*; Schellach, 1907 Mode of infection not known

**History.**—Relapsing fever has been known in America for many years, being recognized as far back as 1844, but it was considered to be identical with the European type until, in 1906, an Englishman who repeatedly travelled between New York and the West Indies was treated by Carlisle in New York for fever which relapsed, and in which spirochætes were found. Novy and Knapp studied these spirochætes and showed that they were distinct from *S. duttoni*.

cular species

erica and in  
In Europe

the cases recorded are due to laboratory infection

**Ætiology.**—The cause of the disease is *S. novy*; Schellach, 1907

six and eight days. The duration of the first attack is about five to six days, and it often begins with rigors, the tongue is moist, except in grave cases, and the jaundice is mild except in severe

uncommonly absent. Usually there is only one relapse, and more rarely two to five relapses.

**Mortality.**—The mortality is not high, varying between 2 and 6 per cent.

**Treatment.**—The treatment is the same as in the European type

## 2 Relapsing Fever of Central America.

Darling has described cases of relapsing fever in Panama clinically similar to the North American type, but the organism has not been definitely classified

## 3 Relapsing Fever of South America.

In South America (Colombia) a relapsing fever occurs in which the spirochaedri resembles more closely *S. duttoni* than *S. noyesi*. According to Robledo this parasite is carried by *Ornithodoros turicata*s

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## 2 Relapsing Fever of Gen

Darling has described cases of rel clinically similar to the North America<sup>1</sup> has not been definitely classified

## 3 Relapsing Fever of South

In South America (Colombia) a relapsin the spirochaudinnia resembles more closely According to Robleto this parasite is c *turicatus*

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## CHAPTER XLIX

### TYPHUS

#### Synonyms.

Gaol fever,

Italian Tif

Exanthema

typhusa, I

**Definition.**—Typhus is an acute specific fever of unknown but probably protozoal cause, spread by the agency of *Pediculus corporis* de Geer, 1778, and characterized by a sudden onset, a maculopetechial eruption, and severe toxæmia, lasting some twelve to fifteen days, and ending in a more or less abrupt lysis

During the eighteenth and nineteenth centuries typhus was well known in Europe, but included typhoid and relapsing fevers, from the former of which it had to be distinguished by the researches of H. L. ... work in ... after has

already been described in the chapter on the Relapsing Fevers

For a long time the disease passed unrecognized in the tropics, ... other it ... deny ... for it, ... oid, it ... d the ... known ... erts of

the tropics

Brill's disease appears to be a mild form of typhus fever, attenu-

ated perhaps by environment and improved sanitation. He has failed to produce the disease in monkeys.

It is curious the way in which different epidemics have been reported as being characterized by special features: thus the Serbian epidemic of 1914-15 showed a great tendency to gangrene of the feet while those in Silesia and Ireland have been associated with bronchial and pneumonic complications.

*Causal Agent*.—The next point in the history is the search for the

the blood  
in the p  
also could be infected

In 1910 Ricketts and Wilder in Mexico showed that the virus was contained in the blood serum from which it could be removed by filtration through a Berkefeld candle.

In 1911 Wilder repeated the filtration experiments with confirmatory results but subsequent experiments showed that the control monkey may have been immune. A point subsequently confirmed

is those to which it attacks both in men and animals.

The causation of typhus remains undiscovered though many researches have been made first by Hallier who in 1868 described a fungus as the causal agent and then by Klebs who found bacilli in 1881 and by Mott and Blore who in the same year described minute screw-like motile organisms as present in the blood during life and micrococcal-like bodies found in cells between the muscular fibres of the heart.

Bruhl described a diplococcus in 1899 Balfour and Porter found another diplococcus In 1903 Gottschlich described a *Piroplasma* existing in non motile endocorpuscular and flagellate free forms in cases in Egypt but these parasites may have only been cellular degenerations and Horiachi described a bacillus which he isolated from the feces and urine of Japanese troops which in the war in Manchuria suffered from a disease which was probably typhus  
Krom  
cular  
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■ pleomorphic  
1916 Penfold  
ccus In 1919

Borrel, Cantacuzene Jonesco and Nasha have isolated a capsulated cocco bacillus somewhat similar to one previously found by Pret jetschsky

In 1910 Ricketts and Wilder saw some curious double bodies in the blood of a number of cases In 1913 Hegler and Prowazek saw numerous long round and diplococcal bodies in the leucocytes some time after infection began but not at the commencement thereof These bodies are believed to be the same as those described in 1905 by Rabinowitsch as *Turkischen Reizformen* In addition they observed the forms described by Ricketts and Wilder not merely in the blood but also in polymorphonuclear leucocytes in the exudate of a blister During convalescence the bodies agglomerate in the cells and finally tend to disappear Post mortem trachoma like bodies were found in the endothelial cells of the heart the lung the liver and the kidney They are found in infected monkeys but not in guinea pigs  
small coccoid and diplo

nineteenth day of the illness These bodies are called *Rickettsia prowazeki* Da Rocha Lima 1916 and according to this author are never found in typhus free lice but can be found in lice infected by sucking infected blood then they penetrate into the cells of the alimentary canal of the louse on the fourth to fifth day after an infective feed and multiply therein and do not reach full development until the eighth to ninth day In this situation they are very small shortly elliptical or oval and often lie in pairs Noller in 1916 considers that the aetiological role of this organism is no longer doubtful and in 1917 Foulerton considered that it was probably a

The virus according to these authors consists of minute coccus like

was most certainly it does not cross air spaces, though it seems to be associated with bedding, fomites furniture, and dirt, which causes the suspicion that the agent may be an animal parasite. Moreover, the fact that it only appears in the cool season of the tropics, and its rapid disappearance in the warm season, is also in favour of its transmission by some animal. Its non infectious nature has been proved by Jurgens keeping twenty healthy men confined with twenty typhus patients freed from lice, with negative results, therefore suspicion is aroused that it may be spread by a blood-

In 1904 Nicouze, Comte, and Conseil transmitted the virus from

these observers contracted the disease, and Prowazek died. The further investigations of Rocha-Lima have already been noted, and it only remains to say that he found that lice kept at 23° C did not become infective, and the organism did not develop, but at 32° C the organism did develop, and the lice became infective. These results are in direct contradiction, as regards temperature, to those of Nicolle, Comte, and Conseil, of Ricketts and Wilder, and of Anderson and Goldberger, and directly opposed to the distribution of the disease. Da Rocha Lima believes the virus is passed on to a second generation of lice, of which larvæ produced from eggs laid by a louse six days after an infective feed are themselves infected. The organism will develop in the human and not in the pig louse.

In 1917 Da Rocha Lima pointed out that Ricketts and Wilder, Gavino and Girard, and McCampbell, have found the parasite in human blood, Von Prowazek in leucocytes and himself in blood in smears, and in sections.

Also in 1917 Otto and Dietrich obtained infections best by allowing lice to feed on the fifth to seventh day of the illness, as only 4-5 per cent became infected on the twelfth day, and all are negative after the fall of the temperature. They infected lice from a patient *sine eranthem* and they confirm *Rickettsia*. On the other hand Brumpt is of opinion that this organism in the louse has

Blaisot's recent  
playing an etio  
found in April,  
the disease

Blaisot prepared an immune serum in horses and asses by the inoculation of emulsions of spleen and suprarenal capsules of infected guinea pigs, and tested its curative power on non-immune guinea pigs, in which it prevents the disease if inoculated in the stage of incubation, and stops the fever at the onset and during the first and second day, but later

in maximum doses of 10 c c per diem. In cases in which the

Parisian typhus are immune to the Lunisian strain

*Prophylaxis*—Many observers (Matland, Strong, Hunter, Jackson, Castellani, etc.) have demonstrated in practice the vital

typhus  
know

*Climatology*.—Typhus is essentially a disease of temperate and



in the Serbian epidemic of 1914-15 when nearly one tenth of the population died from the disease

**Ætiology**—The causal agent which is unknown is spread by means of the louse *Pediculus corporis* de Geer 1778. This insect obtains the virus from the blood of a case in which it is present from the fifth to twelfth day but in greatest abundance from the fifth to seventh day and from which it is absent after the fall of the temperature. The louse requires some eight to nine days interval before it becomes infective. It probably remains infective for the rest of its life but it is not certain whether it passes the virus on to the next generation or not. When an infected louse bites a non-immune human being some six to ten to twelve days elapse before symptoms appear. An attack of typhus confers an immunity upon man and susceptible animals. Natural immunity exists in many animals.

With regard to *Rickettsia prowazeki* Da Rocha Lima 1916 Brumpt in 1918 found that it was present in seventy two *P. corporis*

handled them. *Pediculus humanus* has no *Rickettsia prowazeki*. Brumpt's researches tend to show that *R. prowazeki* is not the causal agent of typhus fever while the observations of Arkwright, Bacot and Duncan are favourable to Da Rocha Lima's theory.

Gutaki in April 1917 reported the presence of a spirochete

scarlet fever bodies have been found by Lopez Vallejo in typhus but have nothing to do with either disease. Hort has described peculiar coccoid bodies in filtered blood. Bradford Bashford and Wilson state that they have grown minute bodies similar to those

egin to  
maxi  
Chari  
action  
ascular  
ash in

this situation

The virus can produce immune bodies in infected animals. In man a second attack is rare but has been recorded and relapses have

hæmorrhages.

**Symptomatology—Incubation**—This varies from four to five, to twenty-one days, according to the older views, but the more correct opinion is some four to ten to twelve days

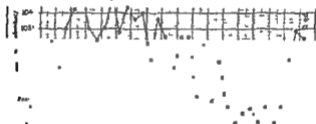


FIG 651—TEMPERATURE CHART OF TYPHUS FEVER



are red, nausea may be experienced, but vomiting is rare, and the bowels are either constipated or normal

dr  
utr  
sometimes an increase in the red cells, with a corresponding increase

malaria or other protozoal infections the polymorphonuclear increase is a characteristic feature, and may exceed 90 per cent., while the mononuclears and lymphocytes may be reduced, especially if the case is to end fatally, while eosinophiles are often entirely absent— a most characteristic feature in a case in the tropics, where worm infection is common. The mononuclear decrease is, however, not so evident in cases about to recover, nor is it present in natives in the tropics, who, of course, are liable to previous protozoal infections, and thus may lower the relative polynuclear count in these regions to 60 per cent. or less.

Though there is sometimes an increase in the red and almost always in the white corpuscles, the specific gravity is said to be lower than normal, which is rather extraordinary, and must indicate, if true, considerable alterations in the plasma. As the disease progresses the rapidity of the pulse increases, and may reach 140 per minute, and is usually small and of low tension. The blood pressure, according to Rizzuti and Scordq, shows nothing characteristic. The respirations are always quickened, and there are generally signs of pharyngitis, bronchitis, or broncho pneumonia, while delirium is not uncommon especially at night.

*The Rash*—Definite preliminary rashes are rare, but there is often very marked flushing of the face, neck, and upper part of the chest, with a *cutis marmorata* or subcuticular mottling of the skin of the lower part of the chest and abdomen. The true rash appears on fourth to fifth day, in the form of roseolar macules, like those seen in typhoid fever, but often more abundant. They are first,

disappear on pressure, and later some of them may slowly fade away, while others, ceasing to disappear on pressure, become petechial, though it is rare for them to develop the dark blue appearance of the petechiæ of such eruptions as those of purpura. This petechial eruption must not be confused with flea bites, which are

and in fatal cases deepens into a profound comatose condition

*Termination*—As a rule the duration of the fever is from fourteen to eighteen days. On or about the fifteenth day the temperature generally falls by crisis or by rapid lysis, which may extend through three to five days; the rash fades, the spleen becomes normal, the leucocytosis increases, and convalescence begins.

In more serious cases the toxæmia may become severe during the

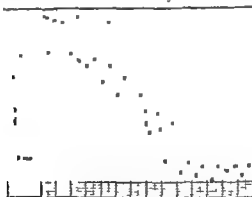


FIG. 657.—TEMPERATURE CHART OF TYPHUS FEVER  
(Chart made by Dr. G. G. Shattock.)

first week, and the patient may die from the seventh to tenth day or even earlier.

*Convalescence*—This may be slow, and not rarely there is danger during this stage, as the general condition may not improve with the cessation of the fever, and death may ensue some two to three weeks after defervescence. In such cases the pulse does not improve, and the patient becomes weaker and weaker until he dies. These are very trying cases for the medical practitioner.

Myocarditis  
pulse  
are  
alignant  
dysentery

*exanthem*—(1) typhus without the rash (5) Typhus in children. This is usually mild, and it is said that death in uncomplicated cases, properly looked after, is rare. Moreover, these cases are im-



negative to Wassermann's reaction. A modification of the Weil Felix reaction with killed  $X_{19}$  is called Neuber's diagnosticum. It is praised by some and condemned by others.

The *differential diagnosis* has to be made from papataci fever, dengue fever, relapsing fever, malaria, enteroidæ fevers, rat bite fever, cerebro-spinal meningitis, pneumonia, septicæmias or

is of the same kind as good for dengue fever

From *relapsing fever* it may be diagnosed by a blood examination showing the absence of spirochætes and the presence of leucocytosis, as well as by the clinical symptoms, but especially the mental disturbance.

From *malaria* it can only be separated by the absence of the malarial parasites which may be present in double infections, and by the leucocytosis.

From *enteroidæ fevers* it may be distinguished by the often abrupt onset, by the leucocytosis, and by the absence of the specific enteroidæ organisms in the blood and fæces.

From *rat bite fever* it may be known by the absence of the mark of a rat bite, absence of the enlarged lymphatic glands, and of the spirochætes in the blood.

apical or basal dulness of the bronchial respiration, as well as by the character of the sputum, with absence of blood.

From *septicæmias and pyæmias* by blood cultures showing an absence of pyogenic organisms, by the absence of blood destruction, and by the presence of cerebral symptoms. From *septicæmic plague* it can be distinguished by the course and blood cultures.

From *uræmia* it is known by the presence of fever and by the analysis of urine.

From *flea bites* the diagnosis, of course, has only to be made

fourth day, absence of shotty papules appearing on the face on the fourth day, but the diagnosis in times of contemporaneous epidemics may be almost impossible. When in doubt and before the specific rash appears, the presence of well marked vaccination marks are in favour of typhus.

From *influenza* by the absence of the catarrhal symptoms.

From *flicæ* by the absence of the buboes and the plague bacilli therein.

To facilitate the diagnosis in cases of typhus with a faint rash Dietsch recommends applying a rubber band round the arm. This procedure makes the rash below the point of application more visible and may cause formation of petechiæ.

the number of the mononuclears, slow pulse, severe petechial eruption or nervous symptoms, alcoholism, pulmonary complaints

it is slightly more fatal in males than in females, while alcoholism, kidney disease, are bad prognostic elements as in a fat or very muscular subject. Pregnant women generally abort, and this complicates the chance of recovery. Complications are generally

jellies, etc., while plenty of water is allowed to be drunk. Careful attention and nursing are required especially when delirious. The temperature should be controlled by cool sponging, and the nervous symptoms by ice to the head, hyocin, bromides or morphine, while the heart is supported by hypodermic injections of strychnine, digitalin, etc. Nicolle finds that the serum of convalescent cases for ten to twelve days after the temperature has fallen to normal has prophylactic and curative properties when given in doses of 20 c.c., repeated if necessary and has manufactured a special horse serum for this purpose. He suggests that in grave

another room, and is sprayed with kerosene oil or petrol, passes into a third room and receives clean or sterilized clothes.

The sterilization of the clothes may be conducted by boiling

#### A. Methods applied to Man —

- I Give illustrated lectures so that people may understand about the louse its habits its association with disease and its prevention
- II Advocate the use of soap and water and of the frequent bath as well as of clean linen frequently changed

B *Methods applied to the louse* —I *Pediculicides* —

them for  
 ously by  
 may be  
 boiled or exposed to steam but must not be in tightly  
 rolled bundles

C *Chemicals* —

*For Use on the Person* —Kerosene oil or petrol spray or vaseline or  
 cresol soap.

*For Use on Stored Clothing* —Naphthalene

II *Lice Repellents* —

Better class patients —Dusting powder of menthol 3-5  
 grains zinc oxide 1 ounce  
 Poorer class patients —Naphthalene as a dusting powder.

III *Special Points* —

He : ?

*Body Lices* —The following drugs arranged in order of effi-  
 cency (according to Castellani and Jackson) may be used —

- 1 Petrol and kerosene oil
- 2 Plain vaseline
- 3 Guaiacol
- 4 Aniso preparations
- 5 Iodoform
- 6 Lysol, cyllin etc
- 7 Carbolic acid, 5 per cent
- 8 Naphthalene
- 9 Camphor

IV *General Insecticides* —

given —

Equal proportions of naphthalene previously soaked in  
 guaiacol or creosote pyrethrum zinc oxide

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## CHAPTER L

# THE SPOTTED FEVER OF THE ROCKY MOUNTAINS

Synonyms—Definition—History—Climatology—Ætiology—Morbidity—Symptomatology—Diagnosis—Prognosis—Treatment—Prophylaxis—The intermittent tick fever of Wyoming—References

### SPOTTED FEVER OF THE ROCKY MOUNTAINS

**Synonyms**—Black fever Blue disease Rocky Mountain spotted fever Spotted fever of Montana Rocky Mountain fever Piroplasmosis hominis Spotted fever of Idaho Tick fever of the Rocky Mountains

**Definition**—An acute endemic febrile disorder associated with a petechial or purpuric eruption of the skin which occurs after the bites of infected ticks *Dermacentor andersoni* Stiles 1905 (which is the same as *D. venustus* Banks 1908) and probably other ticks—e.g. *D. molestus* and *D. maculatus*—in certain regions of the Rocky Mountains

**History**—The first case of the disease is believed to have occurred in Bitter Root Valley in 1873 and from that date until 1902 it is said that about 200 cases were observed with a mortality of 70 to 80 per cent. During this period it was generally known as the black fever the blue disease or the spotted fever.

In 1898 according to Anderson Major M. W. Wood made an unpublished report on the disease to the Surgeon-General of the United States. He called it "Spotted fever" and characterized it as an endemic, non-contagious, febrile disease characterized by severe arthritic and muscular pains and a profuse petechial or



(=*D. andersoni* = *D. venustus* Banks nec Marx) In 1903 Anderson was instructed to investigate the disease, and as a result of his inquiries he supported Wilson and Chowning as regards both the parasite and the tick. In 1905 Stiles published his zoological investigation into the cause, transmission, and source of Rocky Mountain spotted fever, in which he failed to find evidence of the existence of the parasite in man or squirrel, and of the transmission by the tick. His researches were supported in the same year by Ashburn. In 1906 King found distinct experimental evidence of the transmission of the disease by the tick. From 1906 until his recent death Ricketts has been working at the aetiology of the malady and has proved that the tick *D. andersoni* spreads the disease—a conclusion which he has supported by experiments on guinea-pigs and monkeys, but he says that the credit for proving the transmission of the disease from man to man by the tick must be given to McCalla and Brereton. In 1908 Ashburn and Craig published an excellent paper on this and the tsutsugamushi disease which they indicate to be distinct from one another, and in this paper Ashburn accepts the transmission by the tick. Ricketts in 1909 found that there were really two different ticks implicated in the spread of the disease, and these were recognized as *D. venustus* Banks, 1908 = *D. andersoni* Stiles = *D. venustus* Marx, 1897, *pro parte*, and *D. modestus* Banks, of which we have been unable to find a description. It is obvious that there is great confusion

United States  
a, Wyoming,  
New Mexico,

as far as we know, but the causal tick is found there and the fever probably exists therein. In Montana it is found in the Bitter Root Valley on the eastern slopes of the Bitter Root Mountains, and from there to the western bank of the Bitter Root River, by which

June The melted snow drains into the river, which does not  
low water until July. It is only during this period that the ticks

back as 1887. In Oregon it is said to be mild, and to be found only in the eastern portion, towards Idaho.

In 1915 it was noticed at Ismay and Fallow in Montana which was an extension of its distribution. Possibly it occurs in Alaska.

It will be noted that these districts extend from 40° to 47° N latitude and that the elevation is about 3 000 to 4 000 feet above sea level and are sharply defined regions in valleys or at the foot of hills.

Wilson and Chowning noted that the cases occur from March to July as is shown by the following list taken from their report —

March	6 cases
April	24
May	46
June	35
July	5
Spring (exact month not known)	10
Total	126 cases

This seasonal occurrence is associated as stated above with the

Ricketts have failed to find this parasite. Stiles, Ashburn and Crug seem to have thought that the disease was due to a

animal to animal can go on apparently indefinitely (100 generations). These inoculations produce a disease characterized by an incubation a fever an eruption and post mortem appearances similar to those found in human cases. No bacteria can be cultivated from the internal organs or blood of the infected animals but the virus exists not merely in the serum but so closely attached to the corpuscles (white and red) that it cannot be separated from them by washing moreover it will not pass through the pores of a Berkefeld filter. Immunity follows an attack and hyperimmunity can be induced in guinea pigs.

Ricks by centrifuging infected serum for four to six hours at

lymphatics and in the muscle cells of vessels. He thinks that these

organisms may possibly be allied to spirochetes. He has been unable to cultivate them.

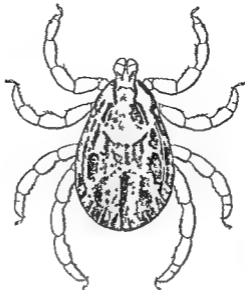


Fig 653 Male



Fig 654 Female

FIGS 653 AND 654—*Dermacentor andersoni* STILES 1905

The virus can be acquired and transmitted by the larva, the nymph, and the male or female adults of *Dermacentor andersoni*.

and in a few instances can pass through the eggs into a second generation of ticks. According to Ricketts the Idaho disease is spread by *Dermatocentor molestus* and the Montana by *D. venustus* Banks 1908 nec Marx 1897 (= *D. andersoni* Stiles 1905). He is inclined to think that there is a difference between the two forms of fever especially as the former has a death rate of some 5 per cent and the latter of about 90 per cent.

Infected ticks are found but sparingly in Nature. Thus of 513 ticks found on animals 296 or more were allowed to attack guinea pigs with the result that only one of the animals took the disease after an incubation of seven days. The infected guinea pig was found to have thirty six male ticks upon it all of which had come from a horse.

A tick fed on a human being suffering from the disease communicated it first to a man and afterwards to a woman by its

animals horses and cattle as well as upon six varieties of wild rodents including *Citellus columbianus* and *Marmota flaviventris* and the jack rabbit but according to Trunks sheep are unsuitable as hosts but this has failed to be confirmed. There is no doubt that the parasite of the fr

opinion that *D.*  
produces a fever  
in Idaho one wit

With regard  
fluences to be noted. Men are more frequently attacked than women and the most common age is from fifteen to fifty years both of which merely signify that persons performing outdoor work run a greater risk of infection than those otherwise employed.

**Pathology** —During the fever the virus can be found in the red and white cells as well as in the serum. It also exists in the liver and spleen.

**Morbid Anatomy** —Rigor mortis is well marked and the skin shows lividity in dependent and petechie in non-dependent parts and at times the marks of the tick bites may still be visible.

The pleura lungs and pericardium and most of the organs are normal but petechie may be seen on the epicardium while the

liver and spleen are enlarged, congested, and soft, and the kidneys are congested, and may show subcapsular hæmorrhages

**Histo-pathology.**—The microscope shows capillary congestion of the organs, with an excess of leucocytes, and an extravasation into and pigmentation of the skin. Acute parenchymatous degenerations of the heart muscle, the spleen, liver, and kidneys, are also to be noted

malaise and nausea, may be experienced

**Onset**—The illness often begins with a distinct chill, accompanied

urine

The fever continues to rise, with slight morning remissions, until a maximum of 105° to 107° F is reached about the fifth to the twelfth day. About the third day (second to seventh) a macular eruption appears on the wrists and ankles which quickly spreads up the arms and legs on to the back, forehead, chest, and abdomen, so that the whole body is included in about one to two days. The macules vary in size from 1 to 5 millimetres in diameter. They are not elevated, and at first disappear on pressure, but later become permanent, and finally turn petechial about the sixth to the tenth day. Associated with the eruption is a dusky red mottling of the skin and often a subicteric tinge of both the skin and the conjunctivæ. The eruption is however, by no means always well marked and, in fact, mild cases have been reported in which it was absent.

The pulse is from the first very rapid reaching from 110 to 150 per minute, and not as a rule in proportion to the temperature. At first full and strong, it becomes gradually feebler and smaller, and is often dicrotic and in severe cases may be intermittent and irregular. The blood shows a diminution of the erythrocytes and hæmoglobin, with a slight increase in the total number of leucocytes and a relative increase of the mononuclear leucocytes, but in considering these blood counts allowance must be made for the altitude at which the disease occurs. Oedema of the face and limbs may be present in severe cases

costal margin, and is tender. There is usually a slight sore throat, and there may be signs of a mild bronchitis associated with an

increase in the number of respirations, which may reach from twenty-six to sixty per minute

The urine presents the usual febrile character, and often contains a trace of albumen, which occasionally may reach considerable proportions, and be accompanied by a few granular casts. More rarely the urine may be scanty or even suppressed. The mind is usually clear, but in severe cases a low muttering delirium may occur.

the fatal event.

*Convalescence*.—Convalescence may take some time, and it is said that the sites of the eruption are clearly visible even twenty-four days after recovery if a warm bath is taken.

*Complications*.—Pneumonia is a comparatively frequent complication, but gangrene of the fingers, toes, and skin of the scrotum and penis may occur. Nephritis, cardiac weakness, and meningitis are possible complications.

*Diagnosis*.—The malady may be difficult to differentiate from typhoid fever, typhus and the Japanese river fever. From *typhoid fever* it can be distinguished by the more acute onset, the petechial eruption, commencing on the hands and wrists, the absence of marked intestinal symptoms, and the presence of leucocytosis.

It is difficult to distinguish between typhoid fever and typhus, though in typhus the disease may end more often by crisis than by lysis, all the other clinical symptoms and the appearance of the eruption are practically identical, in fact, Sambon and others believe that the Rocky

mountain fever, produces pronounced swelling of the scrotum in guinea pigs, and this has been suggested as a test to differentiate Rocky Mountain fever from typhus. Recent immunological studies

**Diagnosis.**—The differential diagnosis from Rocky Mountain spotted fever and typhus has already been discussed (p 1347). At the onset—when the inguinal or other lymphatic glands are enlarged and painful—plague might be suspected. The presence of the necrotic area, and, in any doubtful case, the microscopical examination of the gland juice, which in plague contains numerous bipolar staining bacilli, will enable a diagnosis to be made.

**Prognosis.**—The prognosis is good in the young, and in second and third attacks which are always milder than the first. It, however, gets worse as age progresses, and especially in first attacks. The mortality is about 30 per cent, but increases markedly with age, being only 12.5 per cent in the first, and 57 per cent in the seventh decade of life.

**Treatment.**—Quinine is generally administered, but it does not influence the fever to any marked extent. Salvarsan might be tried. Narcotics may be required to combat the sleeplessness, and constipation must be relieved by purgatives and enemata. Phenacetin, antipyrin, and salicylates are generally badly borne by the patient.

**Prophylaxis.**—The prophylaxis consists in the avoidance of the infective regions during the months of July to October inclusive while the cultivation of the infected regions, and especially the planting of *Eucalyptus globulus* and *Paulownia imperialis*, are advised, as well as the smearing of the exposed parts of the body with eucalyptus oil and balsam of Peru which are said to keep away the mites.

The natives believe that the manuring of the infected lands with human faeces for three consecutive years will make them free from the mites provided there is no flooding during that period.

## ALLIED FEVERS.

### PSEUDO TYPHUS OF DELI SUMATRA

In 1902 Schuffner observed a peculiar fever in Deli, Sumatra, which he described in 1913 and which he thinks may possibly be due to a tick.

Diarrhœa is rare, but pulmonary complications are not unusual, and albuminuria is generally present. The blood shows an increase in the white cells, particularly the lymphocytes, while the eosinophiles are diminished.

No organisms could be found in the blood, and there were no reactions to serum tests for the enteric fevers nor could monkeys be infected by inoculation.

### KOREAN CONTINUED FEVER

A somewhat similar fever to the Sumatra fever is described by Weir. It occurs in spring and early summer. No bite is mentioned, but there is the rash, no diarrhœa, but frequent pulmonary complications and the nervous symptoms. The course of the fever is often short and terminates by lysis.

### MALAY STATES FEVER

Dowden in 1915 described a somewhat similar fever in the Federated Malay States, but gave no ætiological information.

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Jena  
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#### Allied Fevers

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## CHAPTER LII

# RAT BITE AND CAT-BITE FEVERS

Rat bite fever—Cat bite fever—Squirrel bite disease—Addendum—References

### RAT-BITE FEVER

**Synonyms**—Sodoku (So=rat doku=bite) Fièvre par Morsure de rat  
Morso di Topo Rattenbisskrankheit Rattenbeetziekte

**Definition**—A relapsing fever of long duration characterized by redness and swelling at the site of a rat bite often by a generalized papular eruption and caused by *Spiroschaudinnia morsumuris* Gutaki Takaki Taniguchi and Osumi 1916 living in the mouth of *F. pringi norvegicus* Erleben 1777 in many parts of the world and inoculated by means of their bite

**History**—Rat bite fever is mentioned in Japanese medical books from the most ancient times and Scotch French and Spanish literature have references to the disease but it was not until Katsura in 1890 and Miyake in 1899 reported cases that any interest was taken in the malady In 1908 some twenty one Japanese investigators according to Hora had reported some thirty cases which have recently been increased very considerably It has also been recorded by Horder in England by Proescher in the United States in which the literature of the nineteenth century shows occasional cases among settlers by Trugoni in Italy who has given a very good general account of the disease  
Cockin from East Africa As  
first edition of this book there

weasel and it is known that weasels kill rats and therefore it is

observations and experiments of Blake and others<sup>1,2</sup> but is now  
merely of historical interest Douglas Colbrook and Fleming

presence of a spirochete 9-10 microns in length in the lymph glands and in the tissue fluid from the bitten area. Later they found in man and inoculated animals shorter and thicker spirochetes 2-6 microns long, with regular close steep waves and a filament at each end. Ishiwara, Ohtawara and Tamura in 1916 and 1917

† microns and a larger measuring 12 microns in their inoculated guinea pigs.

In 1917 Kaneko and Okuda, in performing a post mortem on a case found —

1 *Long spirochetes*, 6-10 microns in length with numerous small steep, irregular waves, identical with Futaki's long spirochete.

2 *Short spirochetes*, 1-7.5 microns in length with two to six steep close, regular waves identical with the short spirochete of Ishiwara and Futaki, and also to the spirochetes found by Ido and

Hyer. They have also been found in the cortical cells of the supra-

and tissues of infected animals receiving salvarsan treatment.

Ido, Ito, Wani, and Okuda have demonstrated that the serum of persons who have recovered from the disease contains an immune body which destroys *S. morsus moris* as demonstrated by Pfeiffer's test and by the fact that the guinea-pigs employed for this test remain well

purple-coloured eruption and a non-suppurative adenitis following a rat-bite should make the diagnosis easy. The discovery of the

5 per  
caemic  
ions  
For  
seen

ACCOUNTED FOR BY SPOROCHÆTES

**Prophylaxis.**—The rat bite should be thoroughly disinfected.

### CAT-BITE DISEASE.

**Definition.**—A relapsing fever caused by a spirochæte, probably identical with *Spiroschaudinna morsusmuris*, introduced by a cat's bite or scratch.

**History.**—Cat-bite disease was first described in Japan by Fujida and Sato in 1902, while Izumi and Kato in 1903 brought forward

only  
luc

**Ætiology.**—Futaki and Ishihara, Ido, Ito, Wani and Okuda, Izumi and Kato, have all found spirochætes in the blood of patients. They were discovered by the first named and confirmed by the others. The last named believe this spirochæte to be the same as that causing rat-bite, because—

I. The serum of a patient suffering from cat-bite, when mixed with an equal quantity of guinea pig blood containing rat-bite spirochætes, immobilizes them. When repeated with normal and syphilitic serum the spirochætes are not affected.

spirochætes are found. In a control guinea pig the spirochætes were abundant and active.

III. In II the first guinea pig remained healthy and the control died.

**Symptomatology.**—**Incubation.**—This varies from ten to twenty-one days.

**Attack.**—The onset begins with some premonitory symptoms, followed by fever, pains in the muscles and joints, enlargement of the lymphatic glands, and if the skin takes part in the process, carial eruptions.

intervals being  
the disease will

last for months

**Diagnosis.**—This is the same as for rat-bite fever.

**Prognosis**—This is good *quoad vitam* except in old age or debilitated persons

**Treatment**—One injection of salvarsan will cure some cases while others require several injections

### SQUIRREL-BITE DISEASE

**Synonym**—Fiechhornchen Bisskrankheit

Schottmuller described a case of this infection in 1914 in a woman bitten by an African squirrel *Taraxerus cepapi*. The disease was characterized by fever and also by nodules which destroyed the sight of one eye. Another case was a man bitten by the same squirrel and from the pus of this case Schottmuller obtained a nocardia which he called *Streptothrix taraxeri cepapi*. The nocardia infection is probably a complication possibly also introduced at the time of the bite as its presence has been confirmed by numerous observers and species of nocardia are well known to live in the human tonsil and may well exist in the mouth of rats, squirrels and other animals.

### ADDENDUM

Weasels are also said to cause similar symptoms by their bites and it is likely that many other animals do the same and it is possible that they inoculate the bitten person or animal with various types of organisms of which spirochetes appear to be more important as regards the causation of fever.

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917) *T kyō Ijū Sh n hī* (Spara

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## CHAPTER LIII

# THE ENTEROIDEA GROUP OF TROPICAL FEVERS

General remarks—Enteroides or intestinal fevers—Enteric—Parenteric—References

### GENERAL REMARKS.

We use the word *enteroidea*, or intestinal fevers, for all those fevers which are caused by any of the intestinal bacteria, while the term *enteric fever* denotes those which are called *typhoid* and *paratyphoid A and B fevers*. By *parenteric fever* we mean those febrile conditions which though clinically resembling enteric fever, are caused by intestinal bacilli specifically different from *B typhosus* and *B paratyphosus A and B*. These parenteric germs may be closely related to the enteric germs or may be widely separated therefrom (*vide* Chapter XXXVI p 934)

### ENTEROIDEA OR INTESTINAL FEVERS.

The term *enteroidea* covers both the enteric and the parenteric groups and applies to any fever caused by intestinal germs in the widest sense of the word (p 934). These fevers may be classified as follows —

ENTEROIDE FEVERS — Synonyms —Enter oidea Intestinal fevers Enterica <i>sensu lato</i>	}	Enteric Fevers — Synonym Enterica <i>sensu stricto</i>	{	Typhoid Paratyphoid A Paratyphoid B  A due to germs of genus Eber thus and Alcabgenes B due to germs of genus Sal monella C due to germs of genus En teroides D due to germs of genus Lan koides E due to germs of genus Bal kanella and Wesenbergus F due to germs of genus Escherichia
		Parenteric Fevers — Synonym Parenterica	}	

### ENTERIC.

**Synonyms**—Typhoid Fever Abdominal Typhus Gastric Fever Pytho-  
genic Fever Endemic Fever Autumnal or Fall Fever Remittent Fever of  
many writers Common Continued Fever Slow or Lent Fever Nervous Fever

Bilio-gastric  
er  
vre Continue  
éno-meningée

Darmtyphus

The Latin synonyms are very numerous and have been classified by Murchison into —

(a) From supposed resemblance to typhus Typhus nervosus T mitior  
T gangliatus vel entericus Ileo typhus Typhos Typhus (of many old  
writers)

(b) From mode of prevalence Febris non pestilens

(c) From its remittent character Febris semitercianæ seu composita

meenterica magna Febris intestinalis

(i) From supposed origin from worms Febris verminosa Typhus  
verminosus

17 4 002 44 200 — + 2 44 — 17 17

**Definition** —The term enteric fever is used at the present time to indicate three clinically similar fevers, typhoid fever due to *Bacillus typhosus* Eberth 1890, the paratyphoid fevers due to

In the Middle Ages enteric fever, typhus fever, and relapsing fever were always confused with plague until Fracastorius in the sixteenth century distinguished typhus (which included enteric fever and relapsing fever) or febris pestilens from plague or febris vere

pestilens and a febris lenta, the latter being associated with the en

largement of the mesenteric glands · Sydenham, in 1685, described

the term 'febris mesenterica' This fever was characterized by being irregularly remittent, lasting from fourteen to twenty one days, and associated with inflammation of the intestines and mesenteric glands Lancisi thought that the enteric ulcers were caused by round worms Hoffman's febris petechizans vel spuria, Strother's lent fever, Gilchrist's nervous fever, Huxham's slow nervous fever, Riedel's febris intestinalis, and Manningham's febricula, are all synonyms of enteric fever In 1810 Hildenbrand distinguished between contagious typhus and a non contagious nervous fever, which were respectively named typhus exanthemati-

duced the term 'enteric fever' Jenner in 1849 51, completed the clinical differentiation of enteric fever from typhus, from which relapsing fever was also being separated Thus arose the clinical conception of enteric fever, but in the meanwhile many theories had been promulgated as to its causation, thus Bretonneau held the view that it was spread by means of contagion but this opinion was slow in gaining definite support, In 1847 Canstatt pointed out

· · · · · of one of these upon his excrements and possibly

water be-  
milk  
mesenteric  
- 1884

reac  
Grün  
nature

dilution method as devised by Castellani from the circulating blood, assisted the diagnosis, while the bile enrichment method and the Conrad Drigalski's, MacConkey's, and other media generally aided the investigation of the disease Chantemesse has devised an

ophthlmo-diagnostic method. The diagnosis of a mixed infection has been rendered easier by the absorption test as introduced by Castellani in 1902, which is of use also in the determination of closely allied bacteria. The *B typhosus* has been found to be capable of living for weeks in the spleen, and for years in the gall-bladder, of people who are designated 'typhoid carriers,' as the

The bacilli have been  
earth, dust, fomites,  
of relapses has been

studied by Chantemesse and Widal in 1892, Wright and Lamb in

advised a living, not attenuated, sensitized vaccine

The subject of variation of the *B typhosus* has been studied by  
Tunst, Horsfall, and D. of 12

by Schottmuller in 1901, when he clearly demonstrated his *B*

*typhosus B Schottmuller*

The presence of typhoid fever in the tropics has, even in recent times, been much debated, and apparently the view has been held that it was overlooked by the older tropical physicians. This



year Thomson described enteric fever in New Zealand, and Ripley about the same time in Fiji. In 1865 Massey recognized it in Newera Elyia, in Ceylon, while its occurrence in Trinidad and the

which latter became the so called 'typho malarial fever,' which we

first edition of his book, 'Researches on Disease in India,' stated that typh to have h therein statement, for in 1860 he writes that in 1856 he recognized the level on opinion inter

Another point which prevented the general recognition of enteric malarial, and that swelling and ulcerations of Peyer's patches could take place in these fevers, and the practitioner in the tropics was carefully warned that he was not justified in asserting the existence of typhoid fever from the mere character of the post mortem

could be made

Still more recent is the general recognition of the fact that the disease may be prevalent among the natives, in Ceylon, however, n recog- we been

prevalent in many, if not most, tropical countries, and that it fever is occurs, not merely in Europeans, but in natives also

Ætiology.—Enteric fever is caused by the *B. typhosus* Eberth, *B. paratyphosus* A Schottmuller The description of any manual of bacteriology It is al varieties of

each of these germs, and certain characters have been given in  
 Of the B. typhosus for instance two

*typhosus* C. this term, used by several observers, among whom  
 recently Hirschfeld covers different germs, one of which is sero-

are reservoirs or carriers of the bacillus, and may be called *intestinal carriers*. People who pass the bacilli in their faeces during the period of incubation are called *precocious intestinal carriers*, the patients passing the bacilli during the attack and convalescence are called *acute intestinal carriers*, while people who continue to pass the bacilli in their faeces for a year or less than a year after an attack of enteric fever are called *temporary or transitory intestinal carriers*, and those in whose faeces the bacilli are found after

It is that some of the cases of enteric fever may have been cases of

the bacilli are found in the blood. Now

chronic carriers if precautions are taken to prevent the contamination of the urine during or after micturition

In urinary carriers the *B typhosus* has its habitat generally in the renal pelvis which is usually chronically inflamed or cystic and it has also a secondary habitat in the chronically inflamed bladder but it appears possible that it may also live in recesses in the pelvis of the kidney the bladder the urethra the prostate and the vesiculæ seminales from which bacilli pass into the urine The methods of urine infectivity are the same as fecal infectivity

tinal carriers or in some recess or recesses in the urinary tract from the pelvis of the kidney downwards and in the male in the prostate and vesiculæ seminales in the case of urinary carriers This carrier passes the bacilli into the exterior in the feces and urine and these may in cases of defective hygienic surroundings directly infect air food or drinking water or may be conveyed by fomites or by flies

in the gall bladder but its urinary carriers have so far not been recognized It has however been found in the common house-fly in 1911 by Nicoll Its infectivity and its auto infectivity are well known *B paratyphosus A* is prevalent in India and Ceylon where precocious and acute carriers are frequently met with The germ however during the war has now become common also in Europe It is known that it can live in the gall bladder for a considerable time and pass out in the feces and several outbreaks of enteric fever have been traced to this source

With regard to the general question of carriers the persons most liable to be carriers are first of all those who have had attacks of enteric fever and secondly nurses and other attendants upon these patients while the carrier who is likely to prove most dangerous in spreading the infection is a person who is in any way engaged in the preparation or the handling of food intended for other people to consume because the infection of the hands is a prolific source of infection for healthy people

So far we have merely referred to *Musca domestica* Linnæus as

spread of enteric fever

æces or the urine and can infect food and drink directly or in-

indeed no food or cooking utensil can be considered free from possible infection. Of great importance in the tropics are uncooked green vegetables which are grown in gardens often manured by means of human excrement. Fæcally polluted dust and fæcally

during an outbreak of enteric fever and dysentery in a tropical  
 which on investigation  
 a proof of human fæcal  
 from watering the milk

because water is often added by the seller to the milk of *Bos indicus*  
 and this escapes the notice of the buyer because the milk is much

which the oyster bed itself has become infected with bacilli.  
 Another good instance of the cause of small epidemics is the tracing of the infection by Hamer to the fried fish shops so common in London. But the infection of food materials will not give rise to the sudden widespread epidemic which will arise if the water supply is contaminated. Maidstone in 1896 had 35 000 inhabitants of whom 1 900 persons suffered from enteric fever during the months of September, October and November due to contamination of the town's water supply. In the tropics polluted wells are a prevalent cause of endemic and epidemic enteric fever. The present writers were acquainted with a town in which so-called remittent really

can live attached to particles of dust long enough to contaminate food which in certain tropical regions may become covered with dust

is quite common in all races. The position at the present time is that while certain authorities consider that a considerable number of cases of enteric fever still lie hidden under the terms remittent fever and simple continued fever and perhaps febricula as applied to natives and especially to native children who are not often treated by physicians trained in modern methods still there are others who maintain that there is a partial immunity in some races—e.g. such an immunity is said to exist among the native

a comparatively recent attack of enteric fever in about 6 per cent of a hood that

atypical cases that more than one third of the cases of enteric fever whether among Americans or Filipinos are entirely atypical and cannot be diagnosed without laboratory methods

One-half natives to estimate the actual amount of mild and atypical enteric fever which is occurring and to determine why extensive and destructive epidemics are not more often seen

Our own experience in Ceylon causes us to believe that the disease is very prevalent among the natives of that island and as dangerous among them as in Europeans. When the causes of death in the races of Ceylon were considered by us some years ago it was noted that the total deaths contained the following percentages

Race	Enteric Fever	Simple Fever	Remittent Fever
	Per Cent	Per Cent	Per Cent
European	10.1	1.7	1.5
Sinhalese	7.6	12.0	0.3
Tamils	0.7	17.3	3.8

Added together, these give—Europeans, 13.3 per cent, Sinhalese, 19.9 per cent, Tamils, 21.8 per cent. The incidence of

new arrival is apt to eat too much, to drink too much and, being stimulated by the heat—the bad effects of which at first he does

As regards sex, we have already drawn attention to the apparently special incidence upon women between fifteen to forty five years of age

disease most commonly in the dry season, when it is spread by dust and flies, and the natives in the wet season, when it may be due to water contamination

With regard to meteorological conditions, we have observed that anything which prevents the heavy rainfall at the proper season tends to an increase in the enteric, dysentery, etc., rates, and, absurd though it may read, certain observations which we made some years ago led us to believe that the eruption of Mount Pelée may

ditior  
other  
an increased incidence of epidemic intestinal diseases

**Pathology.**—Enteric fever is a septicæmia which is produced by the bacilli already mentioned entering the body by the mouth, and passing into the small intestine and colon, the lymphoid tissue of which they invade. In this tissue they increase in number, and pass via the lymphatics to the abdominal lymphatic glands and spleen, in all of which they multiply. No doubt they very soon reach the blood, but are probably quickly destroyed and their toxins neutralized, and so long as this continues the patient is without definite signs of the disease. This constitutes the period of in-

If the quantity of antitoxin substances produced are only sufficient barely to neutralize the toxins then an ambulatory or an abortive attack may ensue

When, however, the bacilli multiply in such numbers that, though still largely destroyed by the bacteriolysins of the blood, there is insufficient antitoxin to neutralize their liberated toxins, then the fever begins. The possible explanation of the intermittent type of the fastigium of the tropics is that the supply of antitoxic substances in the blood waxes and wanes. The evidence in favour of the above theory is the presence of the bacilli in the fæces in the incubation period, the presence of enlarged Peyer's patches, mesenteric glands and spleen found accidentally in post-mortem examinations of people who have died from other causes and in whom enteric fever was not suspected, the possibility of cultivating

quantity as to produce fever

The bacilli can occur in any part of the body, but they have a predilection for the lymphatic system. When arrested in the lymphatics of the skin, they give rise to the rose-coloured maculo-papules so characteristic of the disease. The appearance and distribution of these red maculo-papules in the skin of the anterior abdominal wall, chest, and back is believed by Greenhalgh to agree with the cutaneous distribution of the nerves which supply the

It has already been noted that they can live for years in the gall-bladder and the pelvis of the kidney, producing the chronic intestinal and urinary carriers, who are liable to infect, not merely themselves but others also.

Auto infection may possibly be the explanation of such cases as have second or even third attacks, within a short period after the first attack.

The typical gradual onset of the disease may be explained by the struggle between the antitoxins of the body and the bacterial toxins.

The occasional sudden onset seen especially in the tropics may be due to lowered resistance, owing to many causes—*e.g.*, climatic influences, other infections etc.

Immunity is generally acquired after an attack, but second



disease most commonly in the dry season when it is spread by dust and flies and the natives in the wet season when it may be due to

tends to an increase in the enteric dysentery etc rates and absurd though it may read certain observations which we made some years ago led us to believe that the eruption of Mount Pelée may  
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Relapses can be explained as being due to any cause which so disturbs the metabolism of the body that the antitoxin production decreases and the germs again gain entrance to the blood in such quantity as to produce fever

The endotoxin contained in the bacilli has a markedly stimulant effect upon endothelial cells causing them to swell and to block small lymph capillaries thus causing patches of focal necrosis in the liver

The bacilli can occur in any part of the body but they have a predilection for the lymphatic system. When arrested in the lymphatics of the skin they give rise to the rose coloured maculo papules so characteristic of the disease. The appearance and distribution of these red maculo papules in the skin of the anterior

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Immunity is generally acquired after an attack but second

Moreover the bowels will usually be seen to be distended with gas and the feces

site of disease is as already stated in the ileum near the ileo-cæcal valve, while the contents of the bowel will be noted to be of a yellowish colour unless there has been a hemorrhage when blood will be seen or unless medicines have been administered which alter the colour of the motions. The Peyer's patches in the lower few feet of the ileum will be enlarged prominent and whitish in colour and covered perhaps with yellowish sloughs or perhaps containing ulcers which may be in the form of one large central or several small ulcers.

On inspection a typical ulcer will be noted to have its long axis in the same direction as the long axis of the bowel to be of oval form with thin and undermined edges and a base formed from the muscularis mucosæ the infiltrated submucosa or from the muscular or even the peritoneal coats of the bowel while in cases of perfora-

patches will be seen to be intensely congested and red in colour. This congestion may be traced for a considerable distance along the ileum and into the jejunum but it is rare to find the duodenum or

be varied by finding only one or  
 1 perhaps only one or two small  
 be no signs beyond a catarrhal  
 inflammation of the mucosa of the bowel. On the other hand the

and almost or quite gangrenous

with a tense capsule while the liver may also be enlarged and con-  
 gested and may even on rare occasions show multiple abscesses  
 brought about by a septic pylephlebitis. The gall bladder and

congested with a capsule which strips off readily. On section both

cortex and medulla are seen to be congested with often fatty degeneration and more rarely small abscesses or infarcts. The

and in the lung pneumonia hæmorrhagic infarcts pyæmic abscesses and purulent infarcts may be seen. The heart is usually flabby and without any sign of rigor mortis and is often pale soft and friable from fatty degeneration. Rarely will vegetative or ulcerative endocarditis or aortitis be found.

In the neck the thyroid gland may in cases of great rarity be

with

an  
 m  
 vitreous degeneration may be found especially in the adductors of the thigh the rectus muscles of the abdomen the pectoralis and the diaphragm and very rarely one of these degenerated muscles may be found ruptured and surrounded by hæmorrhages. The bone marrow may also be congested and show signs of focal necrosis.

Finally there may be the signs of the complications or sequelæ—as for example the arthritis of the joints the abscesses in various

the Peyer's patches.

At first the Peyer's patches are normal in size and number.

Such is the condition of the follicle about the eighth to the tenth day and now one of two things may happen either the excess of lymphoid cells undergo fatty degeneration and absorption with the result that the blood flow returns and the follicle becomes normal or the blockage of the bloodvessels is increased by fibrinous thrombosis with the result that the superficial portion of the swollen follicle dies and forms a slough which separating from the edges towards the centre becomes an ulcer some time during the

second week. These ulcers may increase in depth by an extension of the necrosis and may lead to perforation on the other hand

may also be found at times

The spleen early becomes hyperæmic and swells considerably remaining enlarged until the third or fourth week. The capsule becomes tense and the pulp assumes a dark red colour. The swelling is due to the hyperæmia and cellular infiltration with leucocytes endothelial cells and macrophages among which the typhoid bacilli may be found. During the third week the pulp becomes

#### THE SPECIFIC DEMENT

The kidneys show cloudy swelling of the cells of the convoluted tubules but in the cases when the kidney is specially involved there may be considerable hyperæmia together with perivascular cellular exudation and granular degeneration of the cells of the convoluted tubules.

The heart muscle may show fatty or rarely waxy degeneration while endarteritis obliterans in the small arteries is said to be seen in cases of sudden death without obvious cause.

In the nervous system there are no very marked changes but pigmentation of the ganglion cells and leucocytic infiltration of the perivascular spaces may be seen as well as fatty degeneration of the nerve fibres.

The bone marrow is generally congested and may show signs of focal necrosis with hyperplasia of lymphoid cells and clumps of typhoid bacilli.

death or convalescence. In general terms it may be stated that the fever presents physical signs and symptoms not unlike those seen in the Temperate Zone but a number of cases are atypical. These atypical cases may have a slight and short attack of fever

can disease the  
ubation period  
terminations in

presenting but few symptoms, and very liable to be overlooked, or the attack may be ushered in by some other fever—as, for example, malaria or dengue. We will first describe a typical attack with mild or severe symptoms.

*Incubation*—The incubation period of enteric fever ranges in its known extremes from three days to twenty-three days, or much longer. The shortest known period—that is to say, the one with three days incubation—was exceptional, the infection being due to swallowing a culture of virulent bacilli, and therefore it may be excluded for ordinary purposes. It is by no means easy as a rule to define the incubation period, and it is usual to agree with Murchison and to state that it is most commonly about two weeks (ten to fourteen days), but that it is often less than this, and may possibly be as short as four or five days, while on the other hand, it is often longer, and no definite maximal limit can be mentioned, because

the period of incubation

We therefore conclude that the usual incubation period for enteric fever is about two weeks.

During this period the patient may apparently show no signs or symptoms of the disease, though at times headache and general malaise may be felt.

*Onset*—Typically the onset is gradual, the patient attending to his ordinary duties, though suffering more markedly from the feeling of malaise and lassitude and from headache than during the period of incubation and associated with these symptoms there

medical attendant, when it is found that the temperature is raised above normal ( $100^{\circ}\text{F}$  -  $101^{\circ}\text{F}$ ).

at night

The onset, however, is often atypical in the tropics, the symptoms

being but little marked and the patient though feeling wretched and ill perseveres with his work and may never consult a doctor until well into the second week of the fever and in some cases may even advance farther in the illness than this before the complaint is diagnosed

respiratory system is specially attacked the signs will be those of bronchitis pneumonia or suggestive of acute tuberculosis. If the alimentary canal is selected there will be symptoms indicating irritant poisoning or for attack they will above remarks do n

the onset but enough has been said to indicate the remarkably

marked by the onset of the fever as described above and often the medical attendant does not see the patient until about the end of the third or fourth day of the illness. In the tropics however the attack is at times ushered in by some other fever—as for example malaria by which its symptoms are more or less masked. It is however possible even at this early stage to observe associated

appear. The signs and symptoms to which we specially refer are the alteration of the temperature chart from one of remittent or intermittent fever in malaria to one of more continued fever with more marked headache slowing the pulse the appearance of diastolic murmurs the signs of abdominal distension the local pains the tenderness and perhaps the gurgling in the right iliac region and

listless and drowsy. Headache noises in the ears and pains in various parts of the body are the marked symptoms of this week while sleeplessness is often another marked symptom. Delirium is however rare. The temperature continues high being usually from 103° to 104° F. in the evening with a remission of a degree

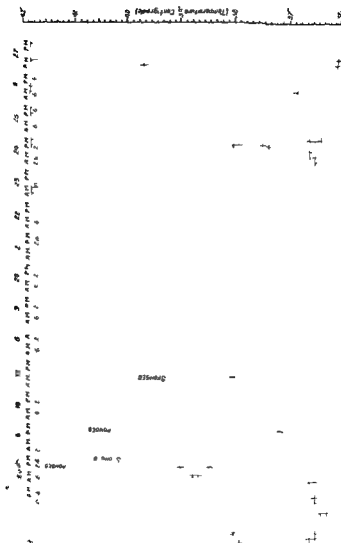


FIG 659A — TEMPERATURE CHART OF THE INTERMITTENT TYPE OF ENTERIC FEVER



first week continue to increase with the exception of the headache

specific bacilli are much more difficult to obtain from the circulating blood but can still be recovered from the spleen the feces the rose spots and often from the urine. Liver or gall bladder symptoms and other complications may appear. With this introduction the signs and symptoms of the second week may be considered in slightly more detail.

During this week the patient who may have been up and about is generally confined to bed and may be seen for the first time by the physician. The facies is dull apathetic and listless the reaction time is prolonged for answering questions the hearing may be diminished and the patient is drowsy. At night there may be sleeplessness and mild delirium. There may be slight sub sultus tendinum. The decubitus is dorsal. The temperature continues high varying from about 102° to 105° F while the pulse is generally relatively slow (90 to 100 beats per minute) although it may reach to 120. The dirotism may have disappeared but the blood pressure has generally diminished somewhat and the first

and toes may be of a bluish colour. The nails often show signs of  $10^{-1}$  of  $+1+$  and transverse ridges may be observed. Phlebotomies persist through the week and pink maculo papules appear in successive crops on the flanks of the abdomen or chest beginning

number and presence depends upon some unknown secondary cause and not upon the severity of the attack to which they bear no reference. They are pinkish or rose red circular slightly elevated

h. no

but rarely met with on the face though they may at times be found on the arms and legs. They continue to appear until the end

# SYMPTOMATOLOGY

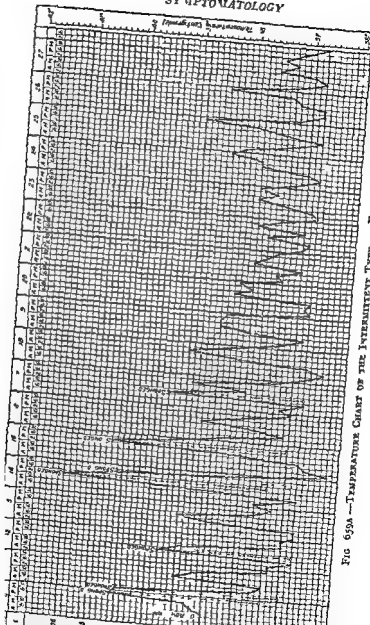


FIG 659A — TEMPERATURE CHART OF THE INTERMITTENT TYPE

of the second week, during the third week, or even during convalescence

The lips and tongue at the commencement of the second week are in much the same condition as at the end of the first week and if the attack is of mild or medium virulence, they may remain in this condition during the whole week but if the attack is severe and if the patient

ance, for in  
through the

with the dark brown scabs, formed from epithelial debris micro-organisms and food which are called 'sordes'. The tongue becomes dry and is covered with a brown or brownish black fur or crust, and may have painful cracks but this condition is largely due to oral sepsis and is not part of the disease and may be more



FIG 659B—TEMPERATURE CHART OF A CASE OF PARATYPHOID A FEVER  
(Chart made by Major Archibald)

continued into the second week as a rule and it is rare to observe vomiting in this week unless there is some complication. There may be constipation or there may be diarrhoea, and the motions are often of a yellowish colour with alkaline reaction and somewhat organized

It varies from a mere trace to a serious hæmorrhage, and most usually occurs from the end of the second to the end of the third week—that is to say during the period of separation of the sloughs

Often there are little or no signs or symptoms to mark a slight

rate or by a slight drop in the temperature. In sudden severe cases of hemorrhage the facies alters becoming pinched and pallid.

the blood shows an anemia due to the reduction of red blood cells while the hemoglobin and the leucocytes are also reduced. The leucopenia is due to a reduction of the polymorphonuclear leucocytes and eosinophiles while the mononuclear leucocytes are usually increased in numbers. The presence of a marked leucocytosis



FIG 659C.—TEMPERATURE CHART OF A CASE OF PARATYPHOID B FEVER  
(Chart made by Major Archibald)

would indicate the occurrence of some complication. The coagulability of the blood is reduced.

Pain and tenderness may occur in the right hypochondrium over

rusty sputum is rare it may be overlooked.

Rarer symptoms during this week are local neuritis tetany eye troubles and ear complications but muscular cramp is by no means rare and may cause great inconvenience to the patient.

The urine is febrile and may contain albumen. The diazo

starvation. In mild cases the fever is usually of the remittent type, but in severe cases it is of the continuous type, and usually the temperature keeps high during the whole week and the patient enters the third week with all the signs and

gradually remit towards the end of the week. In the severer cases the fever continues and the patient passes into a condition commonly called the status typhosus. This is the week in which there is a great danger of hæmorrhage and perforation of the intestine.

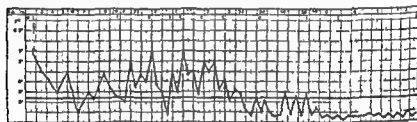


FIG. 660.—TEMPERATURE CHART OF A DOUBLE INFECTION OF TYPHOID AND PARATYPHOID B FEVERS

In the severe cases the temperature remains high, the pulse becomes quick, the tongue, teeth, and lips become covered with sordes, the diarrhoea and abdominal distension become worse, and the toxæmia produces delirium at first mainly at night, but later more or less continuously, or the patient may develop a quiet state of delirium, lying quietly in the bed with the eyes open and staring, and a

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for the pharyngo typhoid ulcer to appear and tympanites develops. The great danger of the third week is perforation, which is said to occur in about 3 per cent of cases. It may take place in the mildest of cases, and we have seen it in one in which only two small ulcers could be found at the post mortem examination, so

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plications may occur

In protracted cases the patient lies in the status typhosus with a high temperature passing urine and feces involuntarily and may die from cardiac failure from perforation or some other complication

*The Fifth and Sixth Weeks* — These should be weeks of convalescence but at any time a relapse may take place while complications and sequelæ may occur

*Varieties* — The various types of typhoid fever may be classified in the following sequence Ambulatory abortive mild typical severe and the masked

The ambulatory is typically presented by a person who feels ill for some days or weeks but goes about his usual work feeling exceedingly wretched until perhaps someone noticing how ill he looks may take his temperature and perhaps find it over  $104^{\circ} F$  thereupon the patient seeks advice for the first time and may be well into the second or even third week of the disease. More rarely a patient may go through the whole attack without medical assistance. Often however the illness may begin with

severe headaches neuralgias early delirium and other marked mental symptoms—e.g. mania or the signs of meningitis. Another example is that in which the pulmonary symptoms are specially marked—e.g. the early bronchitis or the pneumonia. Other examples are the severe gastro-intestinal symptoms imitating

poisoning or the signs of an acute nephritis. Lastly the type in which hæmorrhages begin early and are persistent is often spoken of as the hæmorrhagic type.

One curious form may be just mentioned as for example the spleno typhoid in which the spleen is very markedly enlarged without signs of malaria or relapsing fever.

**Complications and Sequelæ**—The most important complication is malaria but dengue and certain unclassified fevers of intestinal origin are occasionally met with during the first week and complicate the diagnosis. Bedsores and boils are not infrequent complications and loss of hair is a frequent sequel. Venous thrombosis in the femoral vein is a frequent complication and infarction may occur in various organs. Arterial thrombosis is much rarer. Acute ascending myelitis is noted while joints may be attacked giving rise to a typhoidal arthritis and the spine to a typhoidal spondylitis. Periostitis of various bones is not rare. Inflammation of the thyroid gland may also occur. Hæmorrhage and perforation have been noted. Iritis, orbital cellulitis and purulent choroiditis have been recorded as due to typhoid fever but purulent otitis media and mastoiditis described as associated with typhoid fever are generally due to other causes than the typhoid bacilli. Appendicitis and meningitis may also occur.

**Relapse**—One of the most important sequels to an attack of enteric fever is the relapse which may occur at any time during the three or four weeks following the fall of temperature to normal. It usually resembles an ordinary attack of typhoid fever.

**Diagnosis**—The diagnosis of enteric in typical cases is not difficult being based principally on the slow onset of the fever the

has noticed that enteric fever there much more frequently than in

it may be much more enlarged and harder than is usually the case. roseola invisible of course in natives may be absent in Europeans while at times these may present a profuse rash. In a few cases some of the peculiarities met with especially the very irregular type of temperature are explained by the presence of two infections—typhoid and malaria. Individuals who have had an attack of malaria may harbour in their spleen Laveran's parasites for a long time without any symptoms but as soon as the resistance of the organism is diminished by any cause like a chill a disorder in dietetics or the onset of some disease an attack of malarial fever ensues. When these malarial carriers develop enteric the malarial infection breaks out again and probably modifies the course of the temperature. It must be admitted how

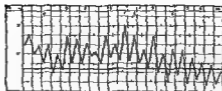


FIG 661.—TEMPERATURE CHART OF A TRIPLE INFECTION OF TYPHOID PARATYPHOID A AND B FEVERS

the tropics. With these clinical difficulties the bacteriological diagnosis of enteric fever acquires in the tropics even more importance than in temperate climates.

The bacteriological diagnosis of enteric is based on the following methods—

- 1 Agglutination test
- 2 Hæmocultures
- 3 Search for the enteric bacilli (*B typhosus*, *B paratyphosus A*, *B paratyphosus B*) in stools and urine
- 4 Splenic puncture



Macroscopic tests (sedimentation test) are also in use, and various

being diluted very little and examined after only a few minutes Dreyer's method, using specially standardized emulsions, is useful, as this method renders much easier the making of agglutination curves, which are of great practical importance in the diagnosis of enteric in inoculated individuals. Technical details may be found in any modern textbook on bacteriology.

In the tropics even at the present time to be placed on the result of practitioners are inclined to be

The practitioner should give his attention to the following points —

*Reaction Negative* — (1) The reaction is generally absent during the first week of the disease

time after inoculation

(4) One must be sure that the test has been carried out with sufficiently diluted blood. A dilution of 1 in 40 is sufficient for ordinary purposes.

**HÆMOCULTURES** — Two methods may be used — the so called 'dilution method,' introduced by Castellani in 1898, and the 'bile enrichment method,' introduced by Drigalski and Conrad, and modified by many authors. The latter is at the present time to be preferred, being simpler. The former is still of use when a mixed

200 or 300 c c of faintly alkaline broth. A dilution of about 1 in 100 to 1 in 200 is generally sufficient. The flasks are incubated at 37° C. Generally, after twelve to twenty four hours in positive cases, the broth becomes cloudy, and shows a growth of the germ.

The germ, of course, must be further identified in the usual way by cultivation in milk, various sugar broths and by the agglutination test. The method gives satisfactory results, the *B. typhosus* having been found by Schottmuller, Auerbach, Widal, Pinot,

titrus are also diluted, and any bactericidal properties of the blood serum greatly weakened.

Gildemeister recommends dilution in sterile water while Cummins and Cumming consider a solution of 0.5 per cent taurocholate of soda to be a very efficient medium.

*Drigalski and Conrad's Bile Enrichment Method*—We use the following modification: 2 to 5 c c of blood are withdrawn from a vein by means of a sterile syringe (see above) or if a vein puncture is absent 2 to 5 c c of blood are drawn from the rectum.

20 c c of the medium. If the blood is taken in the evening the percentage of positive results seems to be larger.

**OPHTHALMO DIAGNOSIS**—Chantemesse has introduced a method of diag-

**COMPLEMENT FIXATION**—A complement fixation test has been worked out for typhoid para A and para B by several authors but its use has not

sown in bile glycerine medium The method gives good results but is not advised as a routine procedure

BACTERIOLOGICAL EXAMINATION OF STOOLS FOR B. TYPHOSUS AND PARATYPHOSE

serum may be added instead of the trivalent one Each tube is inoculated from one of the lactose non fermenting white colonies present on the MacConkey plates made from the suspected faecal matter The tubes are placed in the

... other bacteria DV

ulation add  
drops poly  
etc

apart from enteric group After 24 hours the growth from the top of the tube is further investigated



higher base line of inoculated persons, while there is little or no

If there is a regular rise and later fall in the agglutination titre

1 B para  
s of para  
obability  
all three  
ie patient

suffering from one of the three fevers with non specific agglutininus for one or both the germs producing the other two, or there is the

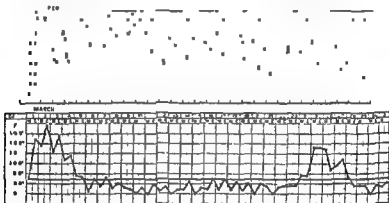


FIG 662—TEMPERATURE CHART OF A TRIPLE INFECTION TYPHOID AND PARATYPHOID A AND B FEVER

possibility of the patient suffering from a mixed infection. In the first case the agglutination curves are generally synchronous, and the germ for which the agglutination is highest is often the infective germ.

In *mixed infections*, whether in inoculated or non inoculated subjects, the agglutination curves for the various infecting germs are as a rule not synchronous, and follow their ordinary course independently of each other.

To carry out the above tests it is necessary to have recourse to an accurate quantitative method, and we recommend for the purpose Dreyer's standard agglutination method, using standard agglutinable emulsions. Details will be found in any modern manual of bacteriology, such as Hewlett's seventh edition.

Of course, in inoculated persons the search for the infecting organisms in the blood, stools, and urine, as already described, is even of

greater importance than in non inoculated individuals; Marris's atropine test may also at times help the diagnosis of enteric in inoculated people, the test apparently not being affected by previous vaccination.

**Differential Diagnosis.**—The term 'enteric fever,' as already stated, is used to cover three infections—typhoid fever, paratyphoid fever A, and paratyphoid fever B, which clinically, at least in the tropics, cannot be distinguished from one another, except by bacteriological methods. Enteric fever imitates many diseases, and many diseases resemble enteric fever, and this renders the differential diagnosis complicated and difficult. Of the many fevers which may possibly be mistaken for some form of enteric fever, the following are the most important:

the fever has lasted a few days, there is generally some sign of anemia. It must, however, be remembered that malaria is a not infrequent complication of the first week of an attack of enteric fever in the tropics, as malarial carriers are very apt to develop this

act  
wh  
reveal the spirochætes

**Dengue**—Dengue begins suddenly with often severe pain in some part of the body, and often congestion of the conjunctivæ, and perhaps a sensation of chilliness. It must not be forgotten that typical cases of dengue fever sometimes develop enteric fever.

**Undulant Fever**—This disease may very closely resemble enteric

type of fever, together with the articular symptoms are characteristic.

**Plague**—The ordinary severe type of plague may be recognized by the sudden onset, the severity of the symptoms, the mental dulness, and the full development of the typical symptoms and bubo in a few hours.

The milder forms of plague are more difficult to recognize, because

form of enteric fever by an examination of the sputum

*Yellow Fever*—Mistakes are most apt to occur at the commencement of a yellow fever outbreak before the disease is recognized. The diagnosis may be effected by the rapid onset of the fever, the tenderness in the region of the pylorus, and by the albuminuria which is often present on the first day, and later by the vomit. But in such an important point as the diagnosis between yellow fever and typhoid fever, bacteriological examination of the blood and

and may be differentiated by the appearance on the fifth or sixth day of a maculo-papular eruption which does not disappear on pressure, and does not appear in crops.

*Influenza*—The diagnosis between influenza and enteric fever can be made by the presence of catarrhal symptoms in the former.

*Trichiniasis*—The presence of oedema of the eyelids, together with swelling and painful tension of the muscles, associated with dyspnoea, are in favour of trichiniasis. The blood should be examined for any signs of eosinophilia while the worms may be discovered in the motions.

*Acute Miliary Tuberculosis*—The differential diagnosis here is very difficult, and is complicated by the fact that the two diseases may occur together. The fever in acute miliary tuberculosis is irregular, and the pulse and respiration are rapid, and there is embarrassment of the breathing, often leading to cyanosis, but the diagnosis is very difficult, and may have to depend entirely upon

usually abrupt, and  
but there are cases  
to be remembered that

symptoms of enteric arise in the second week.

*Rat-Bite Disease*—This may be recognized by the history, by the blotchy, measles eruption, and by the fact that the fever ends for the first time after a few days, only to reappear again later.

*Psittacosis*—This enteric like fever may be suspected by the history of there having been sick parrots in the house in which the

patient has been residing and this can be confirmed by the isolation of the specific bacilli from the blood

*Parenteric Fevers*—These can only be diagnosed by the bacteriological examination of the blood and faeces

*Ulcerative Endocarditis*—Usually in this complaint there are recurring chills irregular fever substernal pains and endocardial murmurs. The blood may not be sterile but the presence of the organisms may indicate the diagnosis

The diagnosis is difficult and depends upon the bacteriological examination but the usually intermittent fever the prostration and the sweats may give rise to suspicion. The marked leucocytosis may also arouse suspicion

*Weil's Disease*—This may be recognized by the early onset of jaundice

- 1 Hæmocultures
- 2 Castellani's absorption test

especially if the blood does not contain specific agglutinins for the germ

*Castellani's Absorption Test*—The usual agglutination tests are not as a rule sufficient to make a diagnosis of mixed infection because

in 1896 demonstrated that the typhoid sera may agglutinate Gaertner bacilli. Zupnik and Poser later found out that 89 per cent of typhoid sera reacted with paratyphoid B and 40 per cent with paratyphoid A.

Boycott, who has made a very complete investigation of the subject has noted that 59 per cent of typhoid sera present co-



agglutinations 55 per cent reacted with Gaertner or Brion and Kayser 41 per cent with Schottmüller B 33 per cent with Aerttrycke and 12 per cent with Schottmüller A

The greater the quantity of typhoid agglutinin the greater as a rule the effect.

to agglutinins and  
inf nisms and mixed  
use fellani in 1902  
and the technique  
improved by Boycott Bambridge O'Brien Cummins and Cummins, Alcock and others. An excellent monograph on the absorption test is the very recent one by Frank T. Taylor.

Castellani found out that in rabbits immunized for typhoid only whose serum agglutinated besides the typhoid bacillus also certain coli germs the saturation with an excess of typhoid bacilli would remove not only the primary (specific homologous) typhoid agglutinin but also the secondary (heterologous non-specific) coli agglutinin while in a serum derived from rabbits immunized both for typhoid and coli bacilli neither saturation with typhoid alone nor coli alone but only both together simultaneously or successively would remove the whole of the agglutinins present in the serum.

He experimented with various other germs and applied the method to the differentiation of closely allied bacilli and mixed infections in man. For practical clinical purposes it may be said

like as the case may be B or paratyphoid A or coli

Of course as with every other biological test the results obtained cannot be accepted as absolute but are only of relative value

should consult books on advanced bacteriology or Taylor's excellent monograph on the absorption test

**Prognosis**—The typhoid mortality is in the tropics about 20 to 25 per cent for ordinary hospital practice and rather lower for private practice. typhoid B is less dangerous than typhoid A. Complications such as hæmic meteorism and relapse is the

Severe hæmorrhages are of grave import as is perforation. Sudden death may occur at any stage of illness and during the convalescence, but is fortunately, very rare.

**Treatment**—The treatment of enteric fever may be divided into—

- A The treatment during the attack
- B The treatment during the convalescence

A **The Treatment during the Attack**—This may be subdivided into—

- I Treatment of a simple uncomplicated case
- II Treatment of special symptoms

I The treatment of a **SIMPLE UNCOMPLICATED CASE** may be considered under the following headings (1) General hygiene, (2) nursing (3) diet (4) medicines.

1 **General Hygiene**—A well ventilated airy and well lighted room should be chosen and all superfluous furniture, hangings, belongings etc. removed except such few things as may be desirable to render the general appearance cheerful. Special attention should be paid to the bed because the patient is to remain in bed for about one month after reaching a permanently normal temperature. The bed should not be too broad or too narrow, and should have a wire-woven mattress which is part of the bed. Over this a soft horsehair mattress should be placed and a reserve mattress should be kept handy. Over the horsehair mattress two folds of blankets should be placed and then the sheet and in the middle third there should be the draw sheet with its water proof sheeting. The bed should be provided with an easily movable mosquito net. The whole room should be thoroughly cleansed once a day by means of damp cloths dipped in Jeyes fluid. All motions and urine should be protected against flies and after being inspected by the physician should be disinfected with Jeyes fluid or crude carbolic acid which is allowed time to act before the contents are thrown away. A separate set of feeding appliances should be reserved for the patient and these should be sterilized after use. All fomites should be soaked in Jeyes fluid or carbolic lotion for some hours immediately after use and before being washed. A large piece of ice is very useful to keep down the temperature of the room in the tropics.

2 **Nursing**—The most important feature of the treatment of a case of enteric fever is the nursing. Two nurses, one for the day and one for the night are absolutely necessary and their work may be rendered easier, and the patient considerably benefited especially if he is over the average weight by the use of a Skeffington lifter or failing this by some simple apparatus based upon the plan of this ingenious lifter. The temperature should be recorded every four hours and as hæmorrhage is so common in the tropics both nurses should be warned and should be instructed to be on the watch for the slightest suspicion of this symptom. Moreover,

they should be instructed how to act when it occurs in order that there may be no delay. The nurse should also be warned to be careful as to the disinfection of her own hands. Nurses who are to attend enteric fever cases should be selected from among those who have been vaccinated, in order to prevent the possibility of infection, failing this a course of intestinal disinfection at the end of nursing a case of enteric fever is not without its benefits as many nurses contract the disease.

th tepid water twice a  
this may be repeated  
xcellent plan is to add  
l 40 grains spirits of

lavender 2 ounces rectified spirits of wine 3 ounces dilute acetic acid 3 ounces in 16 ounces of rose-water. The patient generally finds this admixture to be most refreshing.

From the first the back should be carefully inspected and dried and dusted with a powder composed of boric acid zinc oxide and starch, or some similar powder. Any irritated region should be bathed with rectified spirits.

The mouth must be carefully attended to and a mouth wash of glycothymoline listerine or other antiseptic mouth wash must be used, while the teeth should be carefully cleaned by the nurse by

that the liquid actually given to the patient is chicken broth and

digested or not by examining the fæces. If it is not digested it must be replaced by malted milk zymonized or peptonized milk.

should be given

4 *Medical Treatment*—The less medicine given to a person suffering from uncomplicated and mild enteric fever the better for the patient.

Some mild medicine—*eg* an intestinal antiseptic or quinine in some form—is often given—*eg* the quinine and chlorine made by pouring about 30 minims of strong hydrochloric acid upon 30 grains of chlorate of potash.

mums in a suspension.

Constipation must be counteracted by a simple enema or an enema with a little turpentine if there is some tympanites every other day.

After the second week urotropine may be given in 10 grain doses three times daily in order to disinfect the urine and gall bladder.

*Serums and Vaccines*—Serums have not been successful, some what better results have been recorded by a number of authorities by the use of vaccines. We do not use the vaccine treatment except in some protracted cases with low fever and fairly good general condition.

T h b a n g W a s s e r m a n n

**II THE TREATMENT OF SPECIAL SYMPTOMS**—The special symptoms which require treatment may be considered under the following headings—

- |                 |                   |
|-----------------|-------------------|
| 1 Tympanites    | 7 Delirium        |
| 2 Hæmorrhages   | 8 Cardiac failure |
| 3 Perforation   | 9 Phlebitis       |
| 4 Diarrhoea     | 10 Bedsores       |
| 5 Cholecystitis | 11 Abscesses      |
| 6 Hyperpyrexia  | 12 Bone lesions   |

**1 Tympanites**—Tympanites is to be treated by fomentations, turpentine stupes, by the administration of 15 minims of turpentine every three hours, or 3 to 5 minims of cinnamon oil at the same intervals, or by a hypodermic injection of  $\frac{1}{16}$  grain of eserine.

**2 Intestinal Hæmorrhage**—The nurse should be prepared on the onset of this complication to stop all food and drink except a few sips of cold water, to apply an ice-bag to the abdomen, and to raise the bedclothes on a cradle and to administer either the enema mentioned above, or to give a capsule of 3 minims of turpentine, or both. This is of the utmost importance, as then time is not wasted in getting the treatment under way. When the physician is certain that a perforation has not occurred, a hypodermic injection of morphia is very useful, but this should not be given so as to hide the signs of a perforation. The turpentine capsules may be continued three or more times a day, and calcium lactate in 10 grain doses may also be given.

3 *Perforation*—The only chance is to perform a laparotomy and deal with the perforation surgically but this must be done as soon as possible. After the operation the Fowler Murphy after treatment should be carried out and the patient placed as nearly in an upright sitting position as compatible with comfort. This position is maintained for four days. At the same time a continuous administration of salt solution *per rectum* is carried out and so arranged that the patient obtains 2 to 6 litres per diem for a week and Wainwright's special apparatus for this purpose may be employed.

4 *Diarrhœa*—This may be checked by tannin 10 to 20 grains three times a day or tannigen in the same dose. A very useful adjunct is an enema containing Dover's powder 5 grains tannin 10 grains mucilage of gum 1 ounce and thin starch solution 1 ounce. Bismuth preparations should if possible be avoided as they are apt to obscure traces of blood which may be valuable hints of a possible hemorrhage.

5 *Cholecystitis*—This should be treated by urotropine and when chronic by antityphoidal vaccination or a surgical treatment may be advisable.

6 *Hyperpyrexia* must be combated by tepid cool or even iced sponging by immersion in baths of a temperature between 75° to 85° F. Antipyretic drugs should not be given.

7 *Delirium*—Acute mental symptoms require sedatives or relief of intracranial pressure by lumbar puncture.

8 *Cardiac Failure*—This may require to be combated by hypodermic injections of digitalin or camphor in ether or by strychnine and by saline injections.

9 *Phlebitis*—This usually occurs in one of the legs which must be wrapped in cotton wool after applying ichthyol in linoline (2 per cent).

10 *Bedsore*s—These are usually quite preventable but great care is necessary to dry and to disinfect the back and to harden the skin with spirit lotion. When the sores have developed they should be disinfected twice daily with hydrogen peroxide and a veroform or zinc oxide powder applied. In some cases a protargol ointment (5 per cent) or a balsam of Peru ointment (1 to 2 per cent) are useful.

pain of a typhoid spine and the osteitis or periostitis may require surgical treatment.

B *The Treatment of Convalescence*—This may be subdivided into—(I) The treatment of a simple case (II) the treatment of sequelæ (III) the treatment of the acute carrier.

I *THE SIMPLE CASE*—The most important factor is to keep the patient in bed without any change of diet until twenty one days after the temperature has permanently reached normal and then

to gradually increase and modify the dietary. The patient should not be allowed to sit up or to get out of bed until the temperature has remained normal for about four weeks. In the meanwhile the urotropine treatment should be carried out and if possible a

disseminated sclerosis myelitis etc

III THE TREATMENT OF THE ACUTE CARRIER — If a patient has become an acute carrier he should be treated by antityphoid vaccination as already described and urotropine should also be ad

recourse to surgical measures

**Prophylaxis** — The essential features in the prophylaxis of enteric fever are a pure water supply a good system of drainage and

by it may be stated that  
the results are satisfactory  
an emulsion of typhoid

typhoid monovaccine some infiltration and pain at the point of

were interested in its propagation. The following are the  
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 in 1904 and fir  
 Comm

of the bug as a carrier of the disease

Of great value have been the researches carried out in various colonies by  
 Professor Simpson

Kitasato Teague Galeotti, and others

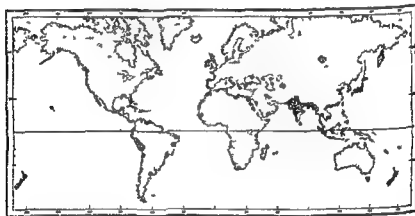


FIG 668.—DISTRIBUTION OF PLAGUE IN 1914

In 1914 plague appeared in Colombo Ceylon where the epidemic was  
 studied by Castellani who isolated the bacillus Pluip and Hirst. Several

discovered

2 Practically all the cases were of the acute septicæmic type and were  
 associated with an extremely high mortality without distinctive features either

the first two are of real importance as far as we know. On the strength of these two different regions an attempt has been made to differentiate two distinct types of the disease: the first the

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often self-l  
cated with  
plague or  
hardly like

disease in the first area is carefully studied. Climate appears to have but little influence on the distribution of the disease and soil apparently none. In India an excessive rainfall seems to favour the spread and virulence of the malady. The hot season of the tropics and the winter season of the Temperate Zone are deleterious to the spread of the disease. The reason for this appears to be the effect of temperature on the bacilli in the flea which disappear rapidly from its stomach above  $85^{\circ}\text{F}$  and are very ineffectual at that temperature while at  $70^{\circ}\text{F}$  they are virulent. Moreover, as has already been stated in the ch...

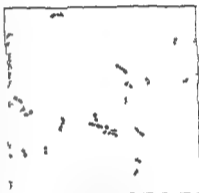


FIG. 669.—PLAGUE BACILLI IN THE BLOOD (X1000)  
(From a photograph by J. J. Bell.)



guinea pigs squirrels mongooses bats jerboas etc it causes the typical disease while in bovines and equines it only causes local reactions while camels are insusceptible Canines birds and reptiles appear to be immune It causes a natural epizootic in rats which apparently is the true disease from which that of man must be looked upon as an offshoot In typical outbreaks there should

bacilli in the bubo the spleen or the blood

The c<sup>1</sup> r<sup>1</sup> m<sup>1</sup> f<sup>1</sup> h<sup>1</sup> d<sup>1</sup>

foci or

the spread of the plague from *R. norvegicus* to *L. rattus*, according to the experiments of the Commission, is neither by direct contact nor by air, soil, or food, but solely by the flea. Contact was excluded by placing healthy rats in the same room with plague-rats which the fleas had been removed, when it

of tangle-foot, overcapable of jumping more than 4 inches, or surrounded by a curtain of wire gauze so fine that a flea could not penetrate it, and exposed to infection,

flea solely with the fæces. It was proved however that the bacilli multiplied in the body of the flea, allowing it to transmit the disease solely on uninfected fleas. The flea becomes infested with abundant bacilli on the twentieth day after infection. In place otherwise much diluted by the feeds with fresh blood. Further it was demonstrated that the disease for seven to fifteen days.

The method of infection probably is in one of two ways—either

1. Directly from the flea into the skin. Martin's experiments tend to show that regurgitation often takes place due to the flea's feeding. Both males and females regurgitate, but that one in ten regurgitates most commonly. The flea is found on rats and the one by which the infection in these experiments

is *Xenopsylla cheopis* with man and because the curve of its epizootic begins to rise ten to fourteen days before that of the epidemic. This period is calculated to be made up of three days during which the flea leaves the dead rat to which is added another three days which is the incubation period of plague in man and five and a half days which is the average duration of the fatal illness in man.

*Xenopsylla cheopis* appears to be the flea by which plague is spread from *Epimys rattus* to man. This rat flea will not merely bite man when it cannot get rat's blood but is capable of living for three to four weeks on man's blood.

*Psylla astia* Rothschild which seems to bite man with great reluctance at temperatures over 80° F.

Pneumonic plague which occurs only in 25 per cent of cases during bubonic epidemics spreads from man to man by bacilli carried by the air for Strong and Teague demonstrated that the

a lot of by people would giant mats of cotton and clothing rags etc

carriers of plague in India having found 22 per cent infected with *B. pestis* when collected from infected native huts. Moreover he successfully transmitted the disease from man to the rat by means of *Cimex rotundatus*.

The possibility of lice acting as occasional carriers should not be forgotten. Lice caught on patients suffering from plague have at

squirrel to squirrel and further that this flea will bite man. Further they record a subacute case of plague in a boy where the infection was believed to be acquired by contact with ground

the disease into three provinces as pneumonic and septicaemic plague while it was spread by the agency of the breath and personal contact of clothes and belongings by coolies travelling in parties and sleeping together in overcrowded insanitary mans especially as the cold of the winter induced an indoor existence. These travelling parties infected the

England, but only a few cases of bubonic plague in man. The tubercle suffers at times from plague, but the bacillus is not

Attention may be called to the fact that the bacillus is not

bacillus

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contain the *Bacillus pestis* in considerable numbers. The bacilli then travel via the lymphatics to the nearest lymphatic glands, which they may traverse, and, passing through the thoracic duct, enter the blood stream, and cause a septicæmia. More usually they remain and grow in the peripheral lymph sinuses of those glands in which they or their toxins cause degenerations of the cells, periglandular serous infiltration, and later, degeneration of the walls of the bloodvessels and hæmorrhage. The lymphatic glands are matted together by the exudation from the primary bubo. The glands usually affected are the femoral inguinal, axillary, iliac, or cervical, which may briefly be classified into the groin, the axillary, and cervical glands.

The Indian Commission points out that the reason why the groin glands are so frequently attacked is simply because they

lymphatics. The bacilli, however, may gain direct access to the blood stream through the injuries to the walls of the veins in the primary bubo. Once the blood stream is reached the disease becomes a septicæmia, and affects the lymphatic glands, forming tertiary buboes, the lungs, causing bronchitis and secondary

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Strong, Crowell, and Turner from a study of the pathology of

inoculations into the lung tissue causing a peribronchial inflammation. From these centres the infection spreads to the adjacent pulmonary tissue and to the visceral pleura the bacilli growing rapidly and causing first a lobular pneumonia and later a lobar pneumonia while the blood becomes quickly infected causing a septicæmia. Secondary pathological changes take place in the spleen, bronchial glands, heart, bloodvessels, kidneys, liver and tonsils which may at times become primarily affected. No sign of any intestinal plague was found although plague bacilli must have been repeatedly swallowed in the bronchial secretions and saliva by the patients.

**Morbid Anatomy**—The characteristic features of a plague post-mortem are the hæmorrhages and the buboes which have been studied in detail by Durck. The skin in the region of buboes

these however, vesicles, pustules or carbuncles may be seen

The secondary buboes are seen to consist of degenerated glands without the œdema but with endo and peri glandular hæmorrhages. The tertiary buboes contain hard hyperæmic glands marked by hæmorrhages.

The muscles of the body, but especially those of the abdominal

and bubo formation. The stomach is hyperemic and hemorrhagic while the solitary glands and Peyer's patches of the intestine are swollen and the mesenteric glands enlarged. The kidney may be normal or show hemorrhages. The nervous system seldom shows any changes but hemorrhages and meningitis may occur.

In cases of *acute septicemic type* there is no post mortem characteristic feature on which the diagnosis of plague may be made or even suspected. The lymphatic glands are not enlarged though the spleen may be of larger size than normal.



FIG. 670.—TEMPERATURE CHART

**Symptomatology**—*Incubation*—The incubation period varies from two to ten days, the average being three days. During this period there may be prodromal symptoms in the form of general malaise, headache

chilliness or rigor, staggering gait, and marked mental dulness.

*Course*—In a few hours the disease becomes properly established when the patient presents a typical facial expression of fear or

rule, however, the temperature is most irregular. Towards the end it may fall by lysis, if recovery is to take place, or fall rapidly to normal or subnormal, and then as rapidly to rise about 107° F. or may simply rise directly to about 107° F. and death ensue. There

disease, and later becoming in bad cases, not countable. At first it is full and of moderate tension, but later it becomes small, weak, and intermittent.

90,000 to 100,000 and more—but in the septicæmic form there may be a leucopenia. The leucocytosis is almost entirely due to an increase in the polymorphonuclear leucocytes.

high and plague bacilli may be found. If hæmorrhages have occurred in the urinary tract, there will be erythrocytes and leucocytes, with albumen and globulin though, apart from this there is often a trace of albumin present, while anuria is generally present before death. Pregnant women always abort.

*Termination*.—As the disease progresses the patient becomes weaker and weaker, the mind wanders, and a wild delirium may occur, but later a low muttering delirium passes into coma and death ensues from the third to the fifth day. In favourable cases the tongue becomes moist and clean, the temperature declines, the pulse rate diminishes, and convalescence begins, but may be much prolonged by suppuration of the buboes or by a secondary septicæmia. Even now death may occur from cardiac failure, suppuration, septic infection, or secondary hæmorrhage.

*Varieties*.—Four variations of the disease are recognized: (1) The ambulatory type, (2) the bubonic type, (3) the acute septicæmic type, and (4) the pneumonic type.



*The Ambulatory Type*—This is the mildest form of the disease and is characterized by but little fever, with slight enlargement and tenderness of a group of lymphatic glands. If the area of skin drained by these glands is carefully investigated a primary vesicle will often be seen at the site of the inoculation and considerably helps in the diagnosis for its fluid contains typical bacilli. The symptoms of this type resemble those already described as prodromata.

*The Bubonic Type*—This type agrees with the general description

diagnosis on clinical grounds may be impossible. Hæmorrhages

hours the temperature is  $103^{\circ}$  to  $104^{\circ}$  F and the pulse 110 to 120 beats per minute. Cough and dyspnœa appear within twenty-four hours when the expectoration is at first scanty but soon becomes abundant. At first it is only composed of mucus but it soon becomes tinged with blood and later very hæmorrhagic and is full of bacilli. The conjunctivæ become injected and the tongue coated with a white or brown and dulness may or may not sounds are usually tubular

heard while the vocal fremitus may be increased. Dyspnœa and cyanosis are early marked features as is the fluid bloody sputum which is found by the second or third day and is full of bacilli. Pleuritis with its usual symptoms may also occur. Prostration is extreme the heart becomes very weak and the pulse rapid while the spleen is usually not palpable. A marked leucocytosis may occur. This is a very fatal type of the disease delirium and coma appear and cases die at the end of sixteen hours two or three or

ess to syncopal attacks  
a general septic infec



at the laboratory the glycerine is wiped off the gland which is made into an emulsion with normal saline and injected half into a guinea pig and half into a rat. It is stated that by this method virulence is maintained for thirteen days which is of great importance in the tropics. A dead animal

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**Prognosis.**—Simpson quotes Procopius of Cæsarea as saying, with regard to the plague in Byzantium that many whose death the physicians predicted recovered, while those who were supposed to be about to recover often quickly died, from which it may be inferred that the prognosis in cases of plague has to be most guarded for dangers are numerous even in convalescence—*e.g.*, cardiac failure.

The prognosis varies with the character of the epidemic, some being milder than others, and is better when the number of cases is declining. It also varies for the type of the disease, being best for the ambulatory and worst for the pneumonic. As 75 per cent of the deaths occur before the sixth day after that time the prognosis improves.

A good pulse, a clear mind, a normal quantity of chlorides and no albumen in the urine, are good signs. High fever, rapid, thready pulse, violent delirium, sudden fall in the temperature, disappearance of the buboes on the fourth or fifth day and anuria, are bad signs. But in all cases be cautious about the prognosis, and remember the physicians of Byzantium. In pneumonic plague the prognosis is most unfavourable—*e.g.*, in the Manchurian epidemic of pneumonic plague no case in which the bacteriological diagnosis was positive was known to recover.

**Treatment.**—The treatment should aim at killing the bacillus and neutralizing its toxins, and naturally one hopes to find those

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be given in large doses (30 to 50 c.c., and even more) by subcutaneous injection. A second and a third injection can be given during the course of the disease but one should keep in mind the possibility of anaphylactic symptoms in people previously in

oculated with serum. Several such cases have been recorded by

nucleated protein from cultures. The mode of action of these sera is said by some not to be bactericidal but to encourage phagocytosis.

Though admitting that the serum treatment is far from being perfect, we advise its use at the very beginning as it gives somewhat better results than the ordinary symptomatic treatment or the so called antiseptic treatment, such as the internal administration of carbolic acid.

As regards the symptomatic treatment, the first indication is to

the wound treated aseptically. Not much good is to be obtained by excising the glands even in an early stage, though several authors—e.g., Terni—have recommended it. The heart's action must be maintained by digitalis, strophanthus, and strychnine. Restlessness may require a hypodermic of hyoscin, hæmorrhage will require treatment by calcium chloride, the so called carbuncles must be treated antiseptically, pneumonia and bronchitis require the ordinary treatment, and if there is constipation, calomel and a saline purge may be given, while vomiting is to be treated as described under Malaria. A sufficiency of fluid should be given, so as to keep up the action of the kidneys.

The diet should consist of broths and milk, while stimulants are usually required.

Tincture of iodine has been recommended, and is given by oral administration a few drops at a time diluted with cinnamon water and may be given also by intravenous injection 5 to 7 minims once or twice daily. Eusol has been recommended by Brayne.

**Prophylaxis.**—*Bacillus pestis* being conveyed by the rat bite from rats to man as well as by the flea, it is

mainly by the flea being carried by human beings on their person,

country from being infected with plague, to institute a Sanitary Service, which should consist of a central authority for collecting

and dealing with information on a large scale, and to which will be attached bacteriologists, who will carry out the necessary bacteriological examinations and prepare prophylactics, and a Port Sanitary Branch, for investigating and disinfecting ships.

It is as well, if possible, to limit the communication with infected areas to as few ports as possible, and to provide isolation hospitals for the sick and for the healthy coming from infected areas. The length of quarantine is fixed at present at five days, but this appears to be too short, if the findings of the Indian Commission with regard to fleas are considered.

Attempts can be made to prevent the disease crossing land frontiers by the medical inspection of persons coming by rail road, or river, but this method of prophylaxis is liable at any time to break down.

It would appear most necessary to disinfect the clothing and persons of all people coming from infected areas as they may carry fleas, and this is most difficult to effect, but it is not so difficult to disinfect merchandise by the Clayton apparatus.

In addition, rats must be caught regularly in places situated in danger zones, and inspected to see if any are becoming diseased for the epizootic begins before the epidemic as a rule.

But of all things the most difficult, as well as the most important, appears to be the recognition of early cases and pneumonia, buboes,

one at the beginning, and the second when the deaths begin to increase rapidly in number. Riots and assassinations of magistrates,

occur, and must be foreseen at a house-to-house inspection be necessary to find out the number of cases, and in no instance must a body be allowed to be buried without being viewed by a medical officer. If this is not done, it will be impossible to gauge the extent of the disease.

Rats must, of course, be regularly and systematically examined, and their mortality curves constructed. The sick must be isolated,

pass from the infected to the non-infected houses, and spread the plague. Village houses might be burnt, which is cheaper and a fairly effective method though, of course, measures should be taken to prevent the rats escaping. The gas in question is generated by burning sulphur at a very high temperature in a special apparatus, when  $\text{SO}_2$  and  $\text{SO}_3$  and some unknown gases are evolved, which are

illness of persons living in places where some sort of virus was being used has been recorded. Danysz's and similar viruses are very often inert and inefficacious in the tropics and require to be exalted by passage through animals before being used.

Bannerman has strongly advised cats as a method of rat extermination but it must be noted that cats are not refractory to plague.

the board on which the bread is cut. The poison which he has used with great success is phosphorus paste. The phosphorus is mixed with glucose to prevent spontaneous combustion, and then a paste is made with a fatty base such as lard but it is advisable to vary from time to time the fatty base. To our minds these little points make all the difference between failure and success. Liston advises the centralization and isolation of the stock of grain in villages and an organization of the system of the refuse disposal, while the stabling of cattle in houses should be prohibited. He lays stress on the disinfection of the clothes of travellers coming from

certainly as well as other insects. One gallon of the 10 per cent solution is sufficient to disinfect a room 12 by 12 in a few minutes; the solution may be washed out afterwards with water. Tobacco and infusions of tobacco will also be found useful as first demonstrated by Castellani and Low when studying the prophylactic measures to be taken against *Pulex penetrans* infections in Africa.

**PERSONAL PROPHYLAXIS**—Ever since the middle of the eighteenth century there has been an idea that a vaccine should be obtained for the purpose of protecting the individual. Wesspremi and later Simonowitz and Cerutti tried artificial inoculation but the results were by no means satisfactory, persons dying from plague as the result of the inoculation. Recently a number of vaccines have been prepared for the purpose of personal prophylaxis which may be classified into (1) Vaccines composed solely of the chemical products of the bacilli (2) vaccines composed of chemical products and dead bacilli (3) vaccines composed of living attenuated bacilli (4) polyvalent vaccines.

**Chemical Vaccines**—In 1897 Lustig and Galeotti prepared a plague vaccine composed of the nucleo-proteids of the bacilli obtained by shaking agar cultures with 1 per cent caustic potash solution and after two hours adding 0.1 per cent acetic acid and thus obtaining a precipitate of nucleo-proteids. It is administered by subcutaneous or intramuscular injections in doses of from 2 to 3 milligrammes. This method has been adopted in La Plata with success and has been experimentally supported by Rowland.

**Vaccines composed of Chemical Products and Dead Bacilli**—The most important is Haffkine's plague prophylactic prepared by growing the bacillus for four to six weeks in broth and then sterilizing at 65° to 70° C for one hour and then decanting into bottles with or without the addition of a little carbolic acid. The dose for an adult man is 3 c.c. for an adult woman 2 to 2½ c.c. and children in proportion to their age (or size). The injection is made subcutaneously into the arm or loin under strict antiseptic precautions. Three or four hours after inoculation the temperature rises and in twelve hours reaches 102° to 103° F while tenderness and swelling may occur at the site of the inoculation and malaise

liquid but it appears more likely that it is in the dead bacilli. Haffkine's vaccine has been very extensively used in India and other countries since several years and on the whole has given extremely good results.

of guinea pigs inoculated intraperitoneally with plague bacilli and killed some little time before they would have died naturally. The exudate is diluted with a solution of carbolic acid and sodium carbonate and chloride and given in a dose of 10 to 25 c.c. By this method plague aggressins are produced and used.

carried out without danger. This vaccination consists of the intramuscular living virus such as cutaneous ten days after the inoculation. Strong has observed a marked rise in the opsonic index and that the phenomenon of complement fixation is given by the blood serum thus proving the presence of

He also prepared and used a pentavaccine plague + cholera + typhoid + para A + para B and an hexavaccine containing in addition Malta fever.

The double

pentavaccine consisted of 1 carbolized saline emulsions of *B. pestis* olea con 00 million was given action was

severe but not more than after Haffkine's monovaccine. The inoculated persons developed immune bodies for the five diseases. The penta- and hexavaccines have recently been further investigated by Castellani and Taylor *Journal of Tropical Medicine* November 1 1917





## CHAPTER LV

# UNDULANT FEVER

Synonyms—Definition—History—Climatology—Aetiology—Pathology—  
Symptomatology—Diagnosis—Prognosis—Treatment—Prophylaxis—  
Para undulant fever—References

**Definition**—Undulant fever is a chronic rarely an acute febrile disorder with many undulatory relapses caused by *Micrococcus melitensis* Bruce 1893 and probably other closely allied germs and usually spread by the agency of goat's milk.

**Remarks**—It seems certain that like enteric, the term undulant fever may cover in reality a group of infections due to very many organisms. Gignaud and Gignaud have described a *Micrococcus* which they have named *M. melitensis* to them Sargent and Zammit have also described a *M. melitensis* which has been proved to differ from that of *M. melitensis* by means of

**History**—Undulant fever has probably existed in the neighbourhood of the Mediterranean for centuries and passages are cited from Hippocrates recounting cases of long drawn out fevers with short apyrexial intervals lasting as long as 120 days which may perhaps refer to the disease.

In the eighteenth and the early part of the nineteenth century references were made to protracted fevers occurring in Malta by various observers—e.g. Howard in 1786 Hennen in 1816 25 Davy in 1842 62—but it is difficult to be certain what disease is referred to. During the Crimean War there appears to have been a very large temporary increase of the fever incidence in Malta much of which was undoubtedly enteric but some of it was not. It is probable that the disease had been imported

from the fever, first gave an accurate account of its clinical history and post mortem appearances under the term Mediterranean remittent or gastric

remittent fever distinguishing it clearly from enteric and what he called Miltese fever

From this date there seems to have been much confusion for it was often diagnosed as some form of rheumatism but the disease appears to have been clearly recognized by Boileau Chirre Thomas and others In 1879 Verle gave an account of it as seen in invalids at Netley as did Gizio in Naples who not merely described the disease but suggested that it might be found to be of bacterial origin

In 1886 it was proved to be a separate pathological entity by Bruce who discovered a micrococcus in the spleen In 1887 he found the organism several times and was able to cultivate it on agar agar and to reproduce the disease by inoculation in monkeys from which he again obtained the organism in pure culture In 1889 he published the first full account of the clinical symptoms



FIG 671 — DISTRIBUTION OF UNDULANT FEVER  
(After Bassett Smith)

and in 1891 he grew the micrococcus from blood aspirated from the spleen during life Thus Bruce proved that this organism which he called *Micrococcus melitensis* was the true cause of the fever

In 1897 Wright and Semple showed that the disease could be diagnosed by the agglutination of the micro organism by the serum of patients and in the same year Hughes published a full historical and clinical account

strated that the micrococcus leaves the body mainly in the urine and is then capable of existing for a long period outside the body Zammit discovered that the milk of many goats agglutinated *M melitensis* and Horrocks isolated the germ from such goats It does not appear to affect injuriously such animals living in the

blood stream and also occurring in the milk by means of which the disease is conveyed to man

A case of paratyphoid fever was reported by Negre and Raynaud in 1912. In 1906

the disease has been reduced by prophylactic measures in some places it had spread to (or been diagnosed in) Spain Portugal and France In the same year Negre and Raynaud described *M para melitensis* and their findings have been confirmed by Bassett Smith who has fully described a case of paratyphoid fever contracted in the South of France

Ceylon Recently it has been reported from many other parts of the world—in fact it may be said to be cosmopolitan and to extend into the interior of the continents A case contracted in Northern Nigeria has been reported by Low

It is certainly a disease of tropical subtropical and temperate climates and exists in addition to the localities mentioned in Russia East and South Africa Uganda where it is called mun hinyo the Sudan Mauretania China the Philippine and Linn Islands North and South America and the West Indies It occurs all through the year but is more common in the warm weather in Malta

Ætiology—The cause as has been indicated in the history is *M melitensis* and some extremely closely allied bacteria *M para melitensis* Negre and Raynaud 1912 and *M pseudomelitensis*

in the expired air, the sweat the saliva or in scrapings from the skin

It can be present in milk and in water fresh or salt for a month, but has never yet been found naturally in air dust soil or

and can live for eighty days in dust or in water fresh or salt for a month, but has never yet been found naturally in air dust soil or

water, neither have any of these in a natural way been proved capable of spreading the disease. It is, however, true that dust

The organism has also been found by the serum reaction to be present in 50 per cent of the goats examined in Malta, and has also been obtained from the milk of 10 per cent of the goats investigated, and, and other a for the ger

It appears to be  
ence in favour of  
which is the infection of a ship's crew by the milk of goats which were being conveyed from Malta to America, 53 per cent of which were found to be infected

The distribution of the disease, therefore, depends upon the con-

suffer, and why occupation and surroundings have but little influence

How the disease spreads from goat to goat is not known. Brumpt states that they drink human and animal urine if deprived of salt and in this way the infection may be kept up. The percentage of infected goats has been found to be 3.4 per cent in Algeria, 29 per cent in St. Marthal, 30.7 per cent in Tunis, 34.2 per cent in Marseilles and 50 per cent in Malta.

Mules, asses, horses, oxen, cows, dogs, rabbits and fowls are all apparently capable of spreading the infection, while often they do not show any sign of disease.

The question of the human carrier is only just coming forward with any degree of prominence as a factor to be considered in the spread of the disease, but Shaw has drawn attention to this possibility in Malta, where many of the dock labourers showed agglutinins in their blood for *M. melitensis* and Missiroli has obtained the micrococcus from the blood of an apparently healthy man who subsequently developed the fever typically in fifteen days.

Four modes of infection are described (1) By the alimentary canal, which is the usual method (2) by the respiratory system, inhaling dust contaminated by goat's urine, which is rare, (3) by

the cutaneous system which is very rare (4) by sexual intercourse which is possible

A variety of *M meli*ensis called *M parameli*ensis is described by Nègre and Raynaud as the cause of a variety of fever termed para undulant fever (see p 1447)

Pathology—The micrococcus enters the blood stream via the

many of its characters without however producing such typical intestinal lesions as that disease. According to some authors (Ross etc) the germ can be conveyed by mosquitoes but this has not been proved. For clinical and experimental reasons Bruce

immune body in the blood suitable for complement deviation and

not merely be congested but the colon may also be ulcerated particularly in cases of hæmorrhage. The lungs are congested,

are usually malaise chilliness headache muscular pains and dyspepsia

Onset—The onset comes on gradually and the patient continues his work though feeling ill while the temperature rises in a ladder being higher each evening and remitting somewhat in the morning. About the fourth or fifth day of the illness a doctor is consulted for the headache and pains in the body and limbs which may have been thought to be rheumatic. The

bronchial catarrh or congestion of the lungs and enlargement and tenderness of the spleen which develop in the course of a week or so

*Course*—All these symptoms continue for about a couple of weeks, 3° to 105° F—but at the end the temperature may become much better. In a day or so, however, a relapse occurs with much the same symptoms as the attack. This relapse subsides, and another follows, relapses and intermissions recurring for months.

The temperature now becomes undulating, with a marked rise at night and fall in the morning, while the patient becomes more and more anæmic, weak, and wasted. The alimentary canal is irritated, as is shown by the dyspepsia and the constipation or diarrhœa. The throat may be sore, the gums spongy, and bleed on pressure, the spleen is often enlarged and painful, and the

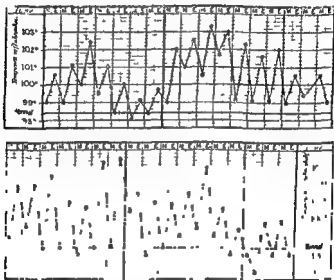


FIG 672—TEMPERATURE CHART OF UNDULANT FEVER

lungs may show signs of bronchitis and occasionally lobular pneumonia, while the heart is easily excited, and hæmic murmurs are heard. The patient shows signs of prostration and head-ache, and pains all over the body are felt. Insomnia and hysterical emotions are not uncommon, but actual delirium is not usual, and the memory may be impaired, while neuritis—*e g*, sciatica—may occur. The skin is pale but damp, due to local or general perspiration, which is very common, and occurs with each remission of the temperature, the sweat being of a peculiar disagreeable odour. Sudamina is not uncommon after the third week. Desquamation may occur, as well as prickly heat, boils, bedsores, subcutaneous abscesses, or hæmorrhages.

The joints may become swollen and very painful, but the skin over

them is seldom reddened. Of all the joints the hip, shoulder, ankle and knee appear most commonly affected. Inflammation or neuralgia of the testes may occur, and at times the parotid also becomes inflamed.

The blood has been more particularly studied by Bassett Smith and Gabb, who find a secondary anæmia with a loss of 20 to 40 per cent of the corpuscles with some poikilocytosis and an even greater reduction of the hæmoglobin. The leucocytic count is normal, but there is a decrease in the polymorphonuclears and an

the specific germ can be found in the urine even two years after an attack. Albuminuria and nephritis may occur, and very rarely hæmaturia.

The patient is now anæmic and prostrated by the repeated attacks of fever, when gradually the symptoms begin to improve.

90 days

Varieties.—Two varieties distinct from the typical description

offensive stools

After a little the symptoms abate somewhat, but instead of improving the pulse becomes intermittent, the breathing is laboured

is usually unassociated with any serious symptoms—in fact so mild may the attack be that it is not discovered until the patient is



general health begins to be unsatisfactory, and he seeks medical advice

*The Ambulatory Variety*—Apparently at times the organism may produce such little effect that the infected person may be unaware that he is suffering from any complaint and may pursue his daily vocation. Thus out of 525 dock hands examined by Shaw at Malta

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mall and  
while persistent vomit  
pleuritis effusion and  
Orchitis is common.

As shown by Trotta the micrococcus may occasionally acquire a pyogenic action and cases of suppurative localized periostitis have been recorded similar to those seen in enteric infections.

*Sequelæ*—Paralysis neuritis common in Cairo wasting of muscles loss of memory, tremulousness and neurasthenia are possible sequelæ

*Diagnosis*—The principal clinical signs on which to base a diagnosis are the prolonged undulant character of the fever the profuse sweatings and the articular symptoms

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be performed in a dilution of at least 1 in 80 otherwise errors in diagnosis will result

(1 in 1000) but in our with a time-limit of thir

for a very long time—two to seven years Birt and Lamb have made this serum reaction the basis of prognosis which is considered to be unfavoura

ing from a high to a low figi saliva (saliva reaction of P

the agglutination test should be carried out in various dilutions to avoid results caused by a paradoxical reaction—viz the possi

Castellani's dilution method as

In case of a negative result the at least three times at different

Nicolle and Conon consider that many of the errors are due firstly to with the clot and state that either the blood

Sasawa and later Missroli and others have applied to Malta fever the rather  
diag

The diseases from which Malta fever must be distinguished are typhoid recognized by a positive Widal reaction malaria recognized by the presence of the blood parasites and kala azar, by its

it as high as 13 per cent for the army in Malta and 9 per cent for the civilian population

Treatment—The treatment is symptomatic as no drug is known which will kill the parasite and vaccine and serum treatments have not been very successful The principal symptoms which

and in the acute stages the bed pan should be used Chills must be avoided by the use of warm clothing and the room should be

cold sponging and ice packs when severe As regards pains

pain

If the colon is affected the treatment may be on the same lines as for a mild attack of dysentery boracic enemata being used Small doses of calomel— $\frac{1}{2}$  grain—three or four times a day as

the nervous symptoms are soothed by cool sponging or by the bromide and morphia mentioned above Insomnia is a common and

water neither have any of these in a natural way been proved capable of spreading the disease. It is however true that dust

animals in food especially milk.

The organism has also been found by the serum reaction to be present in 50 per cent of the goats examined in Malta and has also been obtained from the milk of 10 per cent of the goats investigated and other a for the general infection thus as observed conveyed to which is the infection of a ship's crew by the milk of goats which were being conveyed from Malta to America 53 per cent of which were found to be infected.

The distribution of the disease therefore depends upon the consumption of infected goat's milk for people who use this milk will be more liable to infection than others—hence its occurrence in

explains why there is no age or sex incidence why infants rarely suffer and why occupation and surroundings have but little influence.

How the disease spreads from goat to goat is not known. Brumpt states that they drink human and animal urine if deprived of salt and in this way the infection may be kept up. The percentage of infected goats has been found to be 3.4 per cent in Algeria 29 per cent in St. Marthal 30.7 per cent in Tunis 34.1 per cent in Marseilles and 50 per cent in Malta.

Mules asses horses oxen cows dogs rabbits and fowls are all apparently capable of spreading the infection while often they do not show any sign of disease.

The question of the human carrier is only just coming forward with any degree of prominence as a factor to be considered in the spread of the disease but Shaw has drawn attention to this possibility in Malta where many of the dock labourers showed agglu-

*relitensis* in the urine infecting the soil and in this way may convey indirectly the malady to other persons.

Four modes of infection are described (1) By the alimentary canal which is the usual method (2) by the respiratory system inhaling dust contaminated by goat's urine which is rare, (3) by

the cutaneous system which is very rare (4) by sexual intercourse which is possible

A variety of *M melitensis* called *M paramelitensis* is described by Nègre and Raynaud as the cause of a variety of fever termed para undulant fever (see p 1447)

Pathology --The micrococcus enters the blood stream via the

and may show a glomerular nephritis The alimentary canal may not merely be congested but the colon may also be ulcerated particularly in cases of hæmorrhage The lungs are congested

are usually malaise chilliness headache muscular pains and dyspepsia

Onset --The onset comes on gradually and the patient continues his work though feeling ill while the temperature rises in a ladder being higher each evening and remitting somewhat in the morning About the fourth or fifth day of the illness a doctor is consulted for the headache and pains in the body and limbs which may have been thought to be rheumatic The patient looks ill and his temperature is found to be about 103° F his pulse 80 to 90 and his tongue coated dorsally with a white fur while the edges are red and may be indented by the teeth There is usually a slight sore throat and a tender epigastrium with some bronchial catarrh or congestion of the lungs and enlargement and tenderness of the spleen which develop in the course of a week or so

*Course*—All these symptoms continue for about a couple of weeks to 105° F—but at the end of the first week the temperature may become better. In a day or so however, a relapse occurs with much the same symptoms as the first attack. This relapse subsides, and another follows, relapses and intermissions recurring for months.

The temperature now becomes undulating, with a marked rise at night and fall in the morning, while the patient becomes more and more anæmic, weak, and wasted. The alimentary canal is irritated, as is shown by the dyspepsia and the constipation or diarrhoea. The throat may be sore, the gums spongy, and bleed on pressure, the spleen is often enlarged and painful, and the

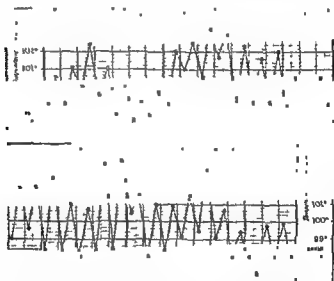


FIG 672—TEMPERATURE CHART OF UNDULANT FEVER

lungs may show signs of bronchitis and occasionally lobular pneumonia, while the heart is easily excited and hæmic murmurs are heard. The patient shows signs of prostration and headache, and pains all over the body are felt. Insomnia and hysterical emotions are not uncommon, but actual delirium is not usual, and the memory may be impaired, while neuritis—*e.g.*, sciatica—may occur. The joints may become swollen and very painful, but the skin over the joints may become red and hot, and sometimes a rash may occur, as well as prickly heat, boils, bedsores, subcutaneous abscesses, or hæmorrhages.

The joints may become swollen and very painful, but the skin over

them is seldom reddened. Of all the joints the hip, shoulder, ankle, and knee appear most commonly affected. Inflammation or neuralgia of the testes may occur, and at times the parotid also becomes inflamed.

The blood has been more particularly studied by Bassett-Smith and Gabbi, who find a secondary anæmia with a loss of 20 to 40 per cent of the corpuscles, with some poikilocytosis and an even greater reduction of the hæmoglobin. The leucocytic count is normal, but there is a decrease in the polymorphonuclears, and an

hæmaturia

The patient is now anæmic, and prostrated by the repeated

90 days

**Varieties.**—Two varieties distinct from the typical description

the other symptoms already mentioned, but in an aggravated form, and often associated with basal pneumonia, and diarrhœa with offensive stools.

After a little the symptoms abate somewhat, but instead of improving, the pulse becomes intermittent, the breathing is laboured, and vomiting becomes serious, and the patient gradually passes into the typhoid state, when hyperpyrexia sets in, and death takes place from the fifth to the twenty first day of the illness.

*The Intermittent Variety*—The onset in this variety is very gradual. When the attack is fully developed, the temperature is

general health begins to be unsatisfactory and he seeks medical advice

*The Ambulatory Variety*—Apparently at times the organism may produce such little effect that the infected person may be unaware that he is suffering from any complaint and may pursue his daily occupation. Thus out of 525 dock hands examined by Shaw at Malta

g diarrhoea hyperpyrexia pneumonia pleuritis effusion and cardiac failure all form serious complications. Orchitis is common shown by Trotta the micrococcus may occasionally acquire a

muscles loss of memory tremulousness and neurasthenia are possible sequelae

**Diagnosis**—The principal clinical signs on which to base a diagnosis are the prolonged undulant character of the fever the

ways resorted to if the blood is inoculated (at least 1 in 50), the test properly performed

diagnosis will result in 1000) but in our with a time limit of three or a very long time—two to seven years. Birt and Lamb have made this serum reaction the basis of prognosis which is considered to be unfavourable if falling from a high to a low figure. Iva (saliva reaction of Polacc

gated for the presence of *A. maitensis* using nitroso nitroastellani's dilution method as in enteric infections (see p 1390). In case of a negative result the examination should be repeated at least three times at different periods of the disease.

Nicolle and Connor consider that many of the errors are due firstly to

Saisawa and later Missiroh and others have applied to Malta fever the Bordet Gengou complement deviation reaction but the technique is rather complicated for routine work

The presence of a high opsonic index for *M. melitensis* may help the diagnosis. A precipitin test has been worked out by Vigano

The diseases from which Malta fever must be distinguished are typhoid recognized by a positive Widal reaction malaria recognized by the presence of the blood parasites, and kala azar, by its parasite in the spleen juice

**Treatment**—The treatment is symptomatic as no drug is known which will kill the parasite and vaccine and serum treatments have not been very successful. The principal symptoms which

and in the acute stages the bed pan should be used. Chills must be avoided by the use of warm clothing and the room should be rendered gnat proof to keep off flies. Care must be taken that the bladder is emptied regularly

The fever is best treated by tepid sponging when moderate and cold sponging and ice packs when severe. As regards pains headache is treated by bromides with or without morphia or small doses of phenazone or phenacetin with caffeine may be used pains in the joints by hot fomentations with belladonna or opium general

dose of calomel and a saline and the bowels kept open by compound liquorice powder or enemata. Diarrhoea is controlled by some ordinary astringent mixture or bismuth or tannalbin powders in some form while Dover's powder is very useful as it also relieves pain

be on the same lines  
enemata being used  
four times a day as  
dyspepsia may require  
a dash of glycothymoline  
or glycerine borax and myrrh or listerine should be used and the throat cleaned by an alkaline spray and then astringed by a gargle. The nervous symptoms are soothed by cool sponging or by the bromide and morphia mentioned above. Insomnia is a common and



distressing symptom and trional and other hypnotics and occa  
 Hemor  
 e careful  
 d calcium  
 chloride The skin must be carefully watched and prickly heat  
 boils or threatening bedsores promptly treated and special care  
 must be taken that after sweating which often occurs at night the  
 clothes are changed The lungs should be watched for signs of  
 congestion and pneumonia or  
 treatment The heart must  
 strychnine iron or some c  
 required

Low diet is necessary during the attack but if milk is used care  
 must be taken that it is not goat s milk or derived from an infected  
 source otherwise while treating the patient a process of reinfection  
 may also be carried out During the intermission the patient  
 should be given light nutritious food care being taken to see that  
 it is really digested

Stimulants in the form of champagne or brandy are often  
 necessary in severe cases because of the cardiac and general  
 depression

De Brum claims to have had good results in the treatment of Malta fever  
 by giving massive doses of quinine (1 drachm daily) Gabbi uses thymol  
 enemata

Scordo recommends intravenous injections of perchloride of mercury while

results in animals with salvarsan

*Treatment by Serums and Vaccines*—Serums have been prepared and used  
 by various authors but the results have been disappointing Vaccines at  
 times give better results especially in protracted cases with low fever and  
 Bassett Smith has recommended that autogenous vaccines in doses of 100-  
 500 millions should be given According to some workers these vaccines give  
 better results when administered intravenously in doses of 25-80 million.  
 Various types of sensitized vaccines have been used and have given at times  
 fairly satisfactory results

**Prophylaxis**—The prophylaxis appears to be simple and to consist  
 in the avoidance of goat s milk The Gibraltar authorities have  
 completely stamped out Malta fever by prohibiting the importation  
 of goats from Malta which together with the diminution of the  
 disease in Malta clearly demonstrates the great practical value of  
 the work performed by the late Commission The average number  
 of cases in the British troops stationed at Malta before 1906 used  
 to be 240 per annum since 1906 condensed milk only is supplied  
 and the number of cases has steadily decreased until in 1910 one  
 case only was recorded It should be kept in mind however that  
 the source of infection is not limited only to the ingestion of con-  
 taminated milk and importance should be given also to direct

infection caused by human carriers and ambulant cases and disinfection of excreta etc should be carefully carried out

The prophylactic measures may be summarized as follows —

*A General Measures*

- (1) Not sit on
- (2) Isolation
- (3) Disinfection
- (4) Sterilization of milk and water
- (5) Good hygiene

*B Personal Measures*

- (1) Personal cleanliness
- (2) Prevention of infection from mother to child
- (3) Vaccination

*C Veterinary Measures*

**PARA-UNDULANT FEVER**

**Definition** — Para undulant fever is clinically similar to or identical

diagnostic tests

**Symptomatology** — From the cases so far reported it is not possible to distinguish clinically between the typical undulant fever and its para variety

**Diagnosis** — This is based upon serological tests

**Prognosis and Treatment** — As for undulant fever

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## CHAPTER LVI

### HEAT STROKE AND HEAT SYNCOPE

Diseases due to physical causes—Heat stroke—Heat syncope—Heat low fever—References

#### DISEASES DUE TO PHYSICAL CAUSES

In Chapters VII (p 137) VIII (p 142) and IX (p 147) of this

and we base our opinion upon the simple experiments which we have given at the commencement of Chapter VII (p 137) and under the section Radiation in Chapter VIII (p 144)

#### HEAT STROKE

**Synonyms**—Sunstroke sun traumatism insolation siriasis (not Sambon's siriasis) thermic fever *French* coup de chaleur coup de soleil *Italian* colpo di sole colpo di calore *German* Hitzschlag Sonnenstich

**Definition**—Heat stroke is caused by a high air temperature especially when associated with marked humidity and is characterized by high fever and often extreme pulmonary congestion convulsions coma and death

**Remarks**—Heat stroke is the form of sunstroke and thermic fever which we have commonly met with in our experience in the tropics

**History**—In the section entitled High Atmospheric Temperatures contained in Chapter VII we have set forth the views of a number of authors with regard to heat stroke sunstroke and heat syncope from early times down to 1908 and it seems to us to be inexpedient to repeat that which we have already written. We will therefore merely continue that history in the following paragraphs. In 1912 and 1913 Hiller made a number of investigations on this subject in the German Army and Fiske in that of the United States while

that there is no difference between heat stroke and sunstroke from a clinical point of view while Rogers and later Bram consider that there is a difference.

Thus Rogers says that the syncopal form is due to the sun and the hyperpyrexial to the heat whereas Bram maintains *inter alia* that sunstroke is associated with a very high temperature and heat stroke with either a normal temperature or a low fever running from 100° to 102° F.

Simpson's experiments as well as our own described in Chapter VII (p 137) show that Haldane's researches detailed in Chapter III (p 62) apply to tropical climates.

In 1915 Puntoni in studying sunstroke made a number of experiments by means of a photographic camera with regard to the penetrating powers of various kinds of rays in which the place of the camera was taken by a piece of the cranium obtained from a post mortem and containing blood. He concluded that the human cranium was diathermal for violet ultra violet rays which he con-

actinic glasses with which we have performed some few experiments in the Sudan and which tested therein are capable of protecting photographic plates.

In 1916 Koizumi as the result of experiments upon an male believed that during severe manual labour in high atmospheric temperatures the products of metabolism are produced in such abnormal amount that they cannot be removed properly and efficiently from the blood and so act as causal agents in the production of heat stroke. This may perhaps help to explain the importance of diet in the prevention of heat stroke.

In 1917 Gruss and Meyer gave an excellent clinical account of an outbreak of heat stroke in Chicago and Amar investigated the effects

to receive moisture therefrom because of its being already sufficiently laden with moisture. They also held that tight and heavy clothing, the ingestion of too little water were minor causes but they were not sure as to the evil effects of alcohol and saw no evi-

monkeys invariably die of heat stroke at a certain time. This helps to prove that the clinical effects of heat stroke can be produced by exposure to the sun as well as by heat in the shade.

He noted that anything which disturbed heat regulation produced fatal results—for example a small dose of atropine did so by disturbing the loss of heat due to perspiration (*vide* Chapter III).

He also observed that intestinal toxins lowered the resistance of the experimental monkeys which is most interesting in view of the fact that man and

of these

It is of course associated with the warm seasons in all countries and with either very high air temperatures in relatively dry climates, or with not so high temperatures if there is much atmospheric humidity.

The disease stands in direct relationship to heat waves as has been pointed out by Rogers in India and well known in temperate climates. Thus according to Gauss and Meyer in July, 1916, the

monthly mean temperature in Chicago was  $78.4^{\circ}$  F., the highest on record, and 152 men and 6 women were admitted to the hospital for heat stroke or heat syncope

*Ætiology* (*vide* also Chapters VI and VII) —The causation of the disease is the action of high air temperatures associated with a

body temperature rises

Haldane's experiments have shown that if the wet bulb thermometer rises to  $88^{\circ}$  F ( $31^{\circ}$  C) in still air, or to  $93^{\circ}$  F ( $34.4^{\circ}$  C) in air to  $78^{\circ}$  F, its appearance is of great interest to a certain

height with a given atmospheric temperature and then remaining stationary, in Haldane's experiments went on rising, and in some measure this corresponded with the temperature of the air

Thus, with reference to the rectal temperature, which is the best indication of the true bodily temperature, Haldane found that with a wet bulb at  $89^{\circ}$  to  $90^{\circ}$  F the rise was  $1^{\circ}$  to  $1.4^{\circ}$  F ( $0.5^{\circ}$  to  $0.75^{\circ}$  C) per hour, at

rest in  
tem

peratures may act as vigorously or more vigorously upon Europeans in the tropics or elsewhere, especially if living under conditions of poor or exhausted health, that

tions in hot countries

1-19

exposure of an animal to a temperature lower than that which would have been fatal. These animals when subsequently exposed to a very high temperature, do show symptoms, but they are less marked, the rise in temperature is not so high the respirations

(especially in the old days of stocks), with a tight belt and knapsack with cross belts, and is at the same time marching in close formation (when the air must certainly be impure from carbon dioxide and dust) under a tropical sun, it is obvious that, especially if there is a fairly high relative humidity in the atmosphere he cannot get rid of this heat, and there is bound to be either thermic fever or heat syncope in a certain number of cases

found in stokers and in soldiers during exhausting marches. Among soldiers the classical instance often quoted is the account of the march of the 43rd Regiment in the Indian Mutiny for over 100 miles mostly across the plains of India in the hottest weather.

The men remained quite well for 969 miles, when they became

There is no doubt that exhaustion and unsuitable clothing were formerly the great causes of the frequency of attacks of heat stroke in the army. In Chicago in 1916 labourers formed 64.9 per cent of the cases but the majority of them were also complicated with alcoholism.

Heat stroke is more frequent, for it is more frequent among Indians, though it is not so common as 8 per cent of the cases in India, so that age may also be a predisposing cause. Previous illness is a great predisposing cause. Hot winds help to induce this condition, in India the hot winds are the main cause of all the cases of heat stroke.

It is well known that animals which a temperature of 45°C kills in one hour, and one of 43°C after a longer lapse of time, the essential pathological change being chromatolysis in the nerve cells, therefore high bodily temperature may cause acute serious nerve-cell changes.

But the effect of high internal temperature has been further investigated by Halliburton and Mott, who have shown that a temperature of 47°C (117°F) is the coagulation temperature of neuroglobulin, while Hewlett had previously shown that egg white would coagulate at a much lower temperature than usual if this is maintained for some time. His experiments were repeated by Halliburton and Mott on cat's brains and they found that at 42°C (108°F) the neuroglobulin separated out but not at lower temperatures. Cat's brains kept at 42° to 43°C. for three and a half hours showed chromatolysis in the nerve cells. They, there-



fore came to the conclusion that a coagulation necrosis takes place in the nerve cells due to the coagulation of the cell globulin and that when this occurs the protoplasm is destroyed

Our own observations entirely agree with those of Marinesco Mott and Halliburton Fig 673 shows a nerve cell in a condition of coagulation from a case of heat stroke. The Nissl bodies, microorganisms, and other inclusions are then that the



FIG 673 —NERVE CELLS FROM THE MEDULLA OF A CASE OF HEAT STROKE SHOWING CHROMATOLYSIS

seat of the lesion of thermic fever is in the cells of the cerebro-spinal nervous system and particularly those of the medulla

Loizum considers acidosis to be the most important pathological finding in thermic fever. He has noted that animals raised on a diet rich in albumin show a higher degree of blood alkalinity and are more resistant to heat stroke

**Morbid Anatomy** —The body retains a high temperature for some hours after death. Rigor mortis comes on quickly and passes off quickly and decomposition on account of the high atmospheric and body temperatures sets in. Lividity is well marked. The

water content for the brain

The organs of the body are in general congested, but especially the lungs, which appear almost black. Some observers have recorded enlargement of the spleen. In our experience there may be enlargement of the spleen, but it has nothing to do with heat

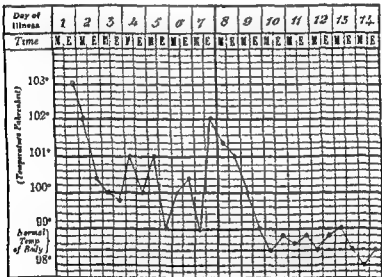


FIG 674.—TEMPERATURE CHART OF A CASE OF HEAT STROKE IN A STOKER WHILE WORKING IN THE ENGINE ROOM OF A STEAMER IN COLOMBO HARBOUR

Temperature 103° F three hours after the onset

stroke. The heart is often in a state of marked rigor mortis, and there may be cloudy swelling of the myocardium as well as in the kidneys and liver. Petechial hemorrhages may be found in the organs and the skin.

of nausea. Longmore lays stress on a frequent desire to micturate as a prodromal sign of importance because, as mentioned by Simpson

nerve symptoms such as restlessness and insomnia. Sometimes these symptoms point to disturbance of the digestive system, in that the patients complain of anorexia, polydipsia, nausea epigastric distress, or diarrhoea. All these prodromal symptoms simply indicate that the patient is not in a normal condition of health.

*Onset*—The attack begins with a sudden sharp rise of temperature

or comatose, usually the latter. The pupils are often very contracted. The respirations are noisy and quick, and râles and rhonchi are heard on auscultation. The pulse is rapid, and though

is much  
is due  
cultures

should be made

*Convalescence*—The patient remains very susceptible to high temperatures for a long time after recovery. There may be definite sequelae.

*Differential diagnosis*—The association with a high atmospheric temperature and a high relative humidity. The differential diagnosis is most important, and has to be made from malaria, epidemic cerebro spinal meningitis, alcoholism, opium poisoning, renal coma, apoplexy, and epilepsy. Heat stroke must be diagnosed from pernicious malaria by the absence of parasites in the presence of Kernig's sign, and of the neck. The high temperature is differentiated from alcoholic or epilepsy (in both of which the rule, between 15 per cent. and 20 per cent. at times. The

Prognosis is therefore always serious and becomes worse if cyanosis or convulsions appear. In the Chicago outbreak 68 out of 158 died and 58 of these never recovered consciousness.

It is too early at present to say how far lumbar puncture will aid the prognosis.

**Treatment.**—Loosen the clothing at once and remove the patient to as cool a place as can be found and take off his clothes and if possible lay him on a bed covered with a large indiarubber sheet, or put him in a bath (care being taken to keep the head supported by some mechanical means so that it cannot slip down into the

circulation in the cooled skin

can be

water  
can be chilled by dissolving a fair quantity of salt in a bath of water and squeezing the juice of some limes into this and then adding a quantity of vinegar and a little eau de-Cologne and sponging the patient with this mixture.

The danger in cold sponging is collapse consequently the rectal temperature must be carefully taken every few minutes and when it falls in the rectum to about  $101.6^{\circ} F$  sponging must be stopped the patient covered up with blankets put to bed and must be most

acted on embarrassed the median basilic vein may be opened and the patient freely bled but if little or no blood comes then an intravenous injection of a pint of normal saline at  $98.6^{\circ} F$  ( $37^{\circ} C$ ) may be run in. Cardiac stimulants such as caffeine digitalis and strophanthus must be employed to keep up the heart's action.

are severe inhalations of chloroform and oxygen mixed may be administered

When the temperature is reduced and the patient is conscious  
 urine can be collected  
 renal trouble which  
 if present, must be treated. Recurrent temperatures may be  
 bacterial in origin and will not be easily reduced but blood cultures  
 should be made and the causal organism determined with a view  
 to vaccine therapy if prolonged

Bromides may be required to soothe the irritated nervous system  
 and do more good than hypnotics or morphine

Food must at first consist of only milk with barley water or  
 toast water and be given in small quantities and often but as con-  
 valescence proceeds broths soups eggs milk puddings etc can

as possible by punkahs and large blocks of ice placed in baths and  
 it must also be kept dark—well protected from the sun's rays  
 is apt to be great  
 thermic fever the  
 climate

An alkaline treatment (ammonium carbonate or sodium bicarbonate) has been  
 recommended with the object of combating acidosis

**Prophylaxis**—Prophylaxis consists in protecting the head and  
 in living in dwellings  
 as cool as possible

the heat of a tropical  
 day if it can be avoided and during these hours alcohol should not  
 be touched

If any of the prodromata are noticed the person should be put  
 on the sick list and treated as though he were ill. He should be  
 kept in a cool shaded room the bowels should be freely opened and  
 cool applications applied to the head. The diet should be light  
 without alcohol and when better the patient should be extremely

trains and the running at the highest speeds possible. Double  
 roofs and windows well protected by coloured glass and jalousies  
 should also be noted as they have been used for years in the Sudan—

## HEAT SYNCOPE.

- 1 High wet-bulb temperatures
- 2 Abnormal bodily health or conditions

The first factor has been sufficiently explained, but a word or two about the second is necessary. The second factor consists of any

temperature

This second factor also includes unsuitable clothing and too violent exercise, which under high wet-bulb temperatures are

generally there is little to note

e  
o

When the temperature is abnormal or there may be a transient initial rise,  $100^{\circ}$  to  $102^{\circ}$  F, and there may be loss of consciousness but this does not always take place. There is often considerable pain in the head.

Generally the condition is quickly recovered from. Sometimes

## HEAT LOW FEVER.

**Definition** —A low intermittent fever of long duration occurring in persons in poor health conditions under the influence of continued high air temperatures and a degree of atmospheric humidity

**Remarks** —The temperature generally rises to about 100° F or less every day for months but the patient may experience little discomfort except that he does not feel very fit or is in a vague way slightly indisposed

**Diagnosis** —It must be diagnosed from low intermittent fever by the fact that in this complaint the patient feels ill

**Treatment** —Rest and change of climate effects a cure for the time being at all events

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## CHAPTER LVII

# THE UNCLASSIFIED FEVERS OF THE TROPICS

General remarks—Cobb's pigmentary fever—Robles fever—Forrest's fever—Naegele's urticarial fever—Hyperpyrexial fever—Double continued fever—Low intermittent non malarial fever—High intermittent non malarial fever—Wossman fever—Nasha fever—Tientsin fever—Whitmore's fever—Woolley's fever with jaundice—The macular fever of  
    n bach—Febris palustris  
    hamocystozoon fever—  
    eat fever—Non malarial  
    fever—Papular fever—  
Hæmorrhagic febrile gastro-enteritis of children—References

### GENERAL REMARKS.

SINCE Crombie in 1898 attempted to arrange tropical fevers much has been done to define these maladies, as may be judged by the preceding chapters. Nevertheless, it is curious to note that the more these fevers are defined and sorted out the greater the number of forms which cannot be classified, notwithstanding a clear definition of *enteroides*.

Hume attempted to arrange these fevers into a typhoid colon

known to us without any system or order

### COBB'S PIGMENTARY FEVER

This latter is peculiar, and reads like a mild attack of insolation. It occurs in the hottest months of the year. The onset is sudden,



**ROBLES' FEVER.**

A peculiar form of continued fever, which is said not to be typhoid and not to be malaria, is described by Robles, of Quezaltenango and Gann, of British Honduras

Robles has separated from the blood of the patients a micrococcus resembling *Micrococcus melitensis*, but liquefying gelatine. The patients, who are usually derived from the younger members of the poorer classes, are anæmic and debilitated and live under insanitary conditions. The fever is very irregular, being at first remittent, but becoming intermittent while the periods of apyrexia increase in length and frequency as the disease progresses. The symptoms are but slight, consisting of headache, malaise, furred tongue, thirst, and anæmia, with slight constipation. The spleen is either not enlarged or but slightly so, while the liver is a little tender on firm pressure. The duration of the disease is from two to three weeks to several months. Convalescence is long drawn out, there being much debility and disinclination for mental or

**FORREST'S FEVER.**

Forrest has described a fever in Rangoon which he called 'Rangoon local fever,' which lasts three to fifteen days, and shows a temperature curve resembling a parabola ascending and descending gradually. The maximum temperature is 104° F, and the blood shows a polymorphonuclear leucocytosis.

**NAEGELE'S URTICARIAL FEVER**

It is found in the tropical regions of South West Africa

due to some food toxin

**HYPERPYREXIAL FEVER.**

**Remarks**—There is a peculiar form of fever which we met with on the Gold Coast and in Ceylon, and which was first described by Thompson and Bennett in Southern Nigeria.

**Climatology.**—It is known to occur on the West Coast of Africa and in Ceylon.

**Ætiology.**—The causation is entirely unknown but peculiar bodies (Fig 675) have been seen in smears from the spleen. We are inclined to consider them contaminations.

Pathology —Nothing is known as to the pathology of the disease

Morbid Anatomy —There is nothing characteristic to be seen in an autopsy

no anxiety is felt about the patient. But on the third day the

temperature begins to rise and reaches  $104^{\circ}$  to  $107^{\circ}$  F, at which it will remain if only drugs are used but if cool bathing is resorted to, the temperature will fall temporarily, rising in due course until cool bathing is again performed. This struggle continues despite any medical treatment that may be employed until at the end of six to seven days the cool bathing ceases to have its effect and the temperature goes on until  $110^{\circ}$  F is reached about the eighth day, and the patient, after having been delirious becomes comatose and dies or, in about 50 per cent of Thompson and Bennett's cases the temperature remains at about  $105^{\circ}$  F for three weeks and then gradually falls to normal about the sixth week.



FIG. 675.—BODIES FOUND IN A CASE OF HYPERPYREXIAL FEVER

The spleen, liver, abdominal organs, urine, and blood appear quite normal except that coagulation is said to be quick. The conjunctivae are injected and the mind is clear until the terminal delirium sets in. The etiology is quite unknown.

Treatment—Cool sponging, cool baths, and cool packs are the only useful treatment.

### DOUBLE CONTINUED FEVER

This disease, which closely resembles enteric fever, was first met with by Manson and subsequently by Thorpe and Roussseau in China.

The disease begins insidiously, the temperature rising to  $104^{\circ}$  F and

are no complications or sequelae.

Treatment must be symptomatic; quinine is useless.

## LOW INTERMITTENT NON-MALARIAL FEVER.

**Remarks**—This fever has been described by Crombie and Castellani. Cases have been reported from India, Ceylon, China, and Siam, where it was observed by Murray. Recently cases have been observed in the south of Italy and the Balkanic zone by Rho and Piebroforte and others.

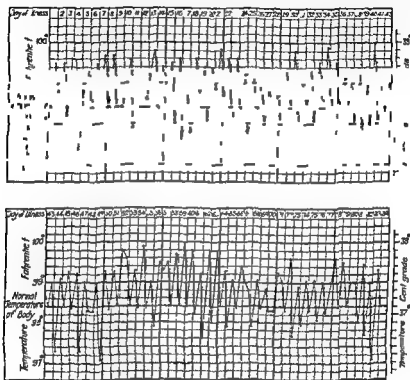


FIG. 676.—TEMPERATURE CHART OF A CASE OF LOW INTERMITTENT NON-MALARIAL FEVER.

**Symptomatology.**—The onset of the disease is insidious or it may follow what to all purposes appears to be a mild attack of influenza.

temperature rises the maximum varying between 99° and 102° F. The temperature never rises in our experience above 102° F. In the evening between eight and midnight, the temperature slowly decreases and becomes normal. Occasionally the patient may perspire when the temperature falls but this is not a constant

symptom. The physical examination of the patient will reveal nothing abnormal, apart from, occasionally, signs of slight anaemia. The liver and spleen are not enlarged. In a few cases some superficial lymphatic glands may be slightly enlarged. In some cases the red blood cells are decreased. An interesting feature in most cases is the distinct increase in the number of eosinophilic leucocytes, even when no evidence of intestinal worms can be found in the motions. The course of the fever is very long. We have seen cases lasting six months.

**Treatment.**—A change of climate is always beneficial, and often quickly stops the fever. A long course of injections of quinine sacodylate is useful in many cases. The ordinary preparations of quinine by the mouth or by injection do not influence the fever in the least.

#### HIGH INTERMITTENT NON-MALARIAL FEVER.

**Remarks.**—This fever has been described by Castellani in 1909 in children in Ceylon.

**Symptomatology.**—Apart, perhaps, from slight anaemia, the child does not show any symptoms except the fever. He takes his food well, runs about and plays, and seems apparently in his usual health. The fever begins in the late morning, and lasts several hours every day, reaching  $103^{\circ}$  to  $104^{\circ}$  F and more. There is no shivering fit at the onset nor perspiration when the temperature falls to normal. The blood does not show anything abnormal, except an increase in the number of eosinophils.

As a

#### MOSSMAN FEVER.

**Symptomatology.**—This fever is characterized by a

and Fielding gave a longer account

### NASHA FEVER.

Synonyms.—Nakra fever, Nakhra Jawhur

Relapses may occur in one to four weeks and sometimes a severe relapse

diminis  
a day  
and

### TIENTSIN FEVER.

A somewhat similar fever has been reported by A. C. Fox from

fourteen days, frontal headache, constipation, slow pulse and constitutional symptoms are slight, relapses are rare, and the mortality is nil while convalescence is rapid. It occurs in persons inoculated against typhoid and having no malaria. It may be enteroides

### WHITMORE'S FEVER.

Synonym.—Morphine injector's septicæmia

of the body. The bacillus isolated seems to be very similar

*B. mallei*.

It is suggested that this fever might exist in the disease is due to the

## WOOLLEY'S FEVER WITH JAUNDICE.

This fever was described by Woolley as occurring in the Andaman

1 cases

40 per

Weeks

disease

## THE MACULAR FEVER OF TUNISIA.

**Remarks**—This fever was described by Conor Bruch and Hayat in 1910 in Tunisia and by other persons more recently

**Ætiology**—The causation is unknown and inoculations into monkeys

## TACAMOCHO FEVER

In 1918 Henao gave an account of five cases of a fever at Tacamocho on the Antioquia Railway Colombia. It was characterized by high fever vomiting intense headache and diarrhœa. The

of yellow fever

## KYOTO FEVER

A fever lasting for seven days in Kyoto Japan and described by Masuda in 1918. Ineda found a spirochete in the blood. The peculiar feature was that adult males of the farmer class were the principal sufferers, and that it produced a cloudiness in the vitreous humour of the eye.

**BAN BACH.**

**Synonym.**—*La miliaire cristalline fébrile.*

This fever was first described by Montel in 1912, and in 1916 by Sarailhé in Cochin China. It is characterized by an insidious onset, followed by six to eight weeks' fever, terminating by lysis and attended by pulmonary catarrh and a very abundant vesicular eruption, which is difficult to see unless looked for, and which comes out in crops. The skin is dry, conjunctiva yellow, and there is constipation, lassitude, and enlargement of the spleen and liver, with rapid compressible pulse and sometimes delirium. Convalescence is prolonged, and there is a complete loss of hair. Blood tests show no parasites, and are negative for the enteric fevers. It is thought to be infectious. This resembles in many particulars the cases de

as to the blood and the feces

**FEBRIS PALUSTRIS REMITTENS.**

Described by Ludwig in 1917 as being characterized by an incubation of twenty one days and a fever of seven to ten days of a remittent type, with headache, pains in the muscles, weakness, jaundice and nephritis. It sounds like *enteroidea*.

**REITER'S DISEASE.**

This was also described in 1917, and resembles the above, but there were pains in the joints, conjunctivitis, iritis and cystitis with enlargement of the spleen and fever lasting about seventeen days.

**OVOPLASMOSIS.**

A fatal fever described by de Raadt in an Annamese aged forty four years, with enlargement of the spleen but not of the liver, and with a temperature somewhat resembling subtertian malaria, without malarial parasites in the blood, but with pigment in the mononuclear leucocytes. Temperature rose very high, and rings were seen stained with any trace of chromatin. These were also seen in the mononuclear leucocytes and were called *Oioplasmia anucleatum* de Raadt, 1913. This sounds like subtertian malaria.

**HÆMOCYSTOZOON FEVER.**

*Hæmocystozoon brasiliense* Franchini 1913, is a flagellate which encysts in the peripheral blood, and was judged to be the fatal cause of a quotidian fever in an Italian physician coming from Brazil.

The fever came on after the removal of a tumefaction in the neck. Spleen and liver were enlarged and there was great anæmia and

without  
 blepharoplast or flagellum (2) Oval or lanceolate forms sometimes dividing  $16 \times 3$  microns (3) Flagellate forms with a flagellum arising near the blepharoplast (4) Non flagellate forms with a large nucleus (5) Encysted forms with chromatinic masses

The patient had enjoyed good health until three years previously when he began to feel ill and lost his appetite while his weight declined. One year later a hard indolent tumefaction appeared on the right side of the neck. This was removed but the wound did not heal and discharged a white non purulent secretion. He now began to suffer from irregular quotidian fever, preceded by

lymphatic glands were normal the urine showed traces of albumen, the blood showed signs of acute anæmia with leucopenia no eosinophilia and a few parasites. Puncture of the liver showed more parasites.

No malarial parasites leishman bodies spores or fungi could be found by examination or by cultures. Wassermann's reaction was negative. Sections of the tumefaction showed granulomatous tissue and some parasites.

### SEPTIC BILIOUS FEVERS

A febrile complaint described by Garton in 1918 as occurring mostly in women associated with enlargement and tenderness of the liver and lasting five to thirty days. One death is recorded.

### BUNGPAGGA.

This appears to us to be myositis purulenta tropica (vide p. 1975)

### ROBB'S HEAT FEVER

This is described as non infective cerebro-spinal fever occurring in East Africa.



### NON-MALARIAL QUARTAN FEVER.

This fever, described by Castellani, is characterized by having a quartan periodicity and no malarial parasites in the blood and quinine given in massive doses does not influence the course, which is prolonged, lasting several months

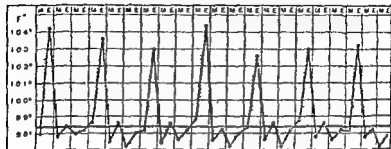


FIG 677—TEMPERATURE CHART OF NON MALARIAL QUARTAN FEVER

### ANÆMIC LOW FEVER.

In young European and native women aged sixteen to twenty two years there is a reaching above 99.4 degree of anæmia become extremely nervous and hysterical This temperature may be due to the degree of chloranæmia and has nothing to do with hysterical fever of certain authors as it disappears on the anæmia being treated

### VESICULAR FEVER.

Definition—A febrile disorder associated with acute pain in

the

temperature

appear at

the com

not related and may on the con

The eruption is generally much less than those  
 There is little  
 first clear and  
 as a rule, no

characters of the  
 eruption and scars  
 as in Jenner's

and often leave

thick crusts

### PAPULAR FEVER

described by Castellani of  
 maculo papular eruption

of this peculiar fever in  
 appearance of the rash but

of the lymphatic glands. The eruption generally lasts between two  
 to five days and then fades without any desquamation and without  
 leaving any marks. In most cases the eruption is associated with  
 itching especially on the arms and in a few cases there is no  
 fever

frankly papular

**Prognosis** —This is good

**Treatment** —The treatment is symptomatic small doses of aspirin or pyramidon being given to relieve the pains and calamine lotion to alleviate the rash.

### HAEMORRHAGIC FEBILE GASTRO-ENTERITIS OF CHILDREN

**Synonym** —Fievre à Vomissements noirs des Enfants

**Definition** —An endemic fever limited to Grande terre in Guadeloupe and characterized by black vomit and by occurring

which resembles yellow fever

The child then becomes very ill with persistent vomiting, slight icterus, obstinate constipation and later black vomit. After lasting from two to five days the symptoms may improve, sleep returns, the fever disappears and the child gradually recovers, on the other hand the symptoms may become worse and the child die. Convalescence is prolonged.

**Diagnosis** —The disease seems to us to be indistinguishable from yellow fever.

**Prognosis** —The prognosis is always grave.

**Treatment** —This is symptomatic.

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P 7

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rd

xvii 129

P 25

**Hyperpyrexial Fever.**

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**Double Continued Fever.**

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**Urticarial Fever.**

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## CHAPTER LVIII

# COSMOPOLITAN FEVERS

General—Epidemic cerebro spinal meningitis—The exanthemata—Vaccination—Alastrum—Vaccinia in natives—Vaccine rashes—Influenza—References

### GENERAL.

25  
ve  
of  
3

### EPIDEMIC CEREBRO-SPINAL MENINGITIS.

**Synonyms.**—Spotted fever *French* Méningite cérébro spinale épidémique

U. S. G. P. O. of the Department of the Interior, Washington, D. C. therefore we

Western Asia and

epidemic

It was first reported in Asia from Smyrna and in 1874-75 in Persia according to Bruce Low in his paper on epidemic cerebro-spinal meningitis published in 1899.

Amen a

In 1874 it was reported in Asia from Smyrna and in 1874-75 in Persia according to Bruce Low in his paper on epidemic cerebro-spinal meningitis published in 1899.

territories of the Gold Coast the natives are said to have noted a great mortality among fowls prior to the epidemic of the disease. According to Pargy and Horn the disease was marked at this time in French West Africa to the west of the Black Volta.

In 1900 it was reported that

since 1906 when Haran noted five cases and has been ably described by Shircore and Ross in 1913

in Ceylon

T. C. L. M. and O. Farrell published a series of investigations upon

bro spinal meningitis first reported in 1897 and Koch's meningococcus and the *Diplococcus pneumoniae* in which it was decided that Biondè's organism was the pneumococcus, but the confusion was increased by Bordoni, Uffreduzzi

separate, or whether there is some relationship between the human and some undefined fraction of the animal complaint

Thus we see that confusion has arisen not only between a disease seen in animals but also between two human organisms



since 1906 when Haran noted five cases and has been ably described by Shircore and Ross in 1913

In 1915 Butler drew attention to a curious feature of the disease in that

*cerebro-spinal meningitis*

in  
ye  
In

in Ceylon

In Ceylon

undefined fraction of the animal complaint

1. In an epidemic von Langelsheim found the meningococci to be present in 23.12 per cent of the cases but as many of the samples came from a long distance it is interesting to note that he obtained 70.6 per cent positive results from examinations in the hospital near the laboratory and taken during the first

1. 1. 1911

mentioned below

In 1908 von Langelsheim gave the following differentiation of the organisms found by him in the naso-pharynx of the contacts —

(1) *Micrococcus catarrhalis* — Frequent colonies dry crumbling when seen under microscope granulated generally with irregular borders. Attacks neither grape sugar nor maltose nor levulose

(2) *Diplococcus flavus I* — Colonies on ascitic agar very similar to those of meningococcus. Twenty-four hours culture exhibits clear yellow pigment in thick layer

(3) *Diplococcus flavus II* — Colonies polymorphic sometimes moist and

meningococcus. Grows also on gelatine at room temperature. According to many authors, markedly pathogenic for mice

(6) *Micrococcus cuneus* — Coarse uneven granular. Colonies and cultures

delicate reminding one of *D. crassus*. Does not attack grape-sugar-levulose and maltose

coccus

- (I) With serum from No 1 19 strains
- (II) With serum from No 20 8 strains
- (III) With serum from No 28 4 strains
- (IV) With serum from No 32 1 strain

They also distinguished by these sera Dopter's parameningococcus from a 1

agglutination tests by devising a simpler method  
(We have now of

mainly because of an experiment in which the meningococcus was found not to cause gonorrhoea when injected into the human urethra

As regards the meningococcus we can aptly quote Crowe who writing in 1915 says —

The present state of our knowledge so far as it bears on the carrier may

certain amount has been written with regard to the so-called pleomorphism of the meningococcus. The meningococcus was first described by Friedl. Thomas and

an attack of the disease, and, moreover, as we have pointed out above, many of the contacts show the meningococcus. It is true that, as a rule, but few attendants on cases acquire the disease, though there are marked exceptions.

It is also true that the accidental infection by Kiefer of his own nose with a meningococcus was observed.

a and Koller and  
ans of subdural  
ebro spinal fluid  
ltures, although

infecting a goat  
ghe and Gryser  
us to be doubt-

out according to  
S

uncertain

Davis in 1905 showed that the coccus multiplied in two normal defibrinated bloods but in four other bloods it was killed as it was by the blood from

up the problem

With regard to the serum treatment Jochmann in 1905 experimented with a specific immune serum which was afterwards manufactured by Merck. He tested his serum by the opsonic test, the bactericidal test and by agglutination.

Kolle and Wassermann also made an immune serum upon which they reported in 1907.

In 1906 F. ...

three deaths and nineteen recoveries or alone and combined with vaccine

conclusions —

covers while symp-  
tom of cerebro-spinal  
the following con

immunity

5 There are various strains of *Neisseria intracellularis*, and to be successful

(a) The search for isolation of and treatment of cases and carriers and the use of vaccine therapy is of use in helping to cleanse cases and carriers

(b) The increase of the immunity of the general population, which can probably be done by prophylactic vaccination in doses of 5, 50 and 100 millions.

scattered patches of pus

In ordinary fatal cases there is considerable amount of purulent exudate in the subarachnoid the most intense being over the uppermost part of the vertex

**ADUULT**

*Attack*—The onset is sudden and may begin with a rigor in an adult or convulsions in a child but these may be absent. The patient feels very ill

stage of the disease only a moderate amount of leucocytes amounting to about 10 000 cells per cubic millimetre will be found while the differential



ing 2 per cent are generally mononuclears. As the day goes on there may be pains in the limbs and some slight catarrhal symptoms in the nose and throat.

*Slight Remission*.—On the second day there is often but not always a slight remission of the symptoms which may be assigned to the treatment but which is really part of the disease.

*Course*.—On the third day if the rash has been ...

may develop but the petechial rash is extremely rare. The leucocytosis is now more marked.

*Terminations*.—During the fourth and fifth days death is not uncommon but the symptoms may persist and death occur later on somewhere about

as time passes on

**Relapses.**—These are not uncommon, and are usually mild in type

**Sequelæ.**—There is usually some permanent damage to the nervous system in cases which recover

be tried

natives it is rather too strong for Europeans

limited

#### THE EXANTHEMATA.

*Scarlet fever* has often been introduced into the tropics but it does not spread. Thus we have seen cases introduced into Colombo from the steamers but there has never been an epidemic of scarlet fever in that town to our

knowledge nor have we seen an indigenous case. The tropical practice

see ere in Ocean a

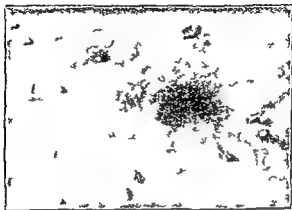


FIG 678—CHICKEN POX FIFTH DAY OF THE ERUPTION

a  
a m t

### VARIOLA

**Synonyms**—Smallpox *Fren* & *La pet te é ole* *Ital an* *Vajuolo* *Grman*  
*Blattern* *Span* & *Viruelas* *Arab c* *Jadart*

ere written. It would appear that the disease was introduced into Cura

t op ca. When first introduced among peoples previously unaffected with the disease it was most virulent—as for example its incidence on the Mex

(p. 533)

cotton workers

in these early stages

The typical rash appears on the second, third or fourth day as papules on

the face arms back of the wrists and hands and later on the trunk and

the tropics—viz. variola hæmorrhagica and varioloid—and these must be mentioned at greater length



FIG 679—SMALLPOX

(From a photograph by Balfour)

**VARIOLA HÆMORRHAGICA**—Hæmorrhagic smallpox is or was by no means rare in Africa and is met with in Asia though by no means so frequently

There are two varieties *Purpura variolosa* and *Variola hæmorrhagica pustulosa*

*Purpura Variolosa*—This variety commences with the same symptoms as

blotches are situate and bleeding occurs from the mucous membranes and internal organs of which the most common are oozing from the gums and

Varicella — The first point in the differential diagnosis between

pricking

From *Varicella* — The first point in the differential diagnosis between smallpox and chicken pox is to remember that it is very difficult and that the most distinguished physicians have owned to not one mistake but a series. No one point of absolute diagnostic value can be given but the following table will indicate some of the points —

<i>Sign or Symptom</i>	<i>Chicken pox</i>	<i>Smallpox</i>
Initial symptoms	Absent as a rule but may resemble smallpox	Usually well marked but may be absent
Temperature	Does not fall with the appearance of the rash	Falls with the appearance of the rash
Situation of rash	Most marked on the trunk	Most marked on the face and limbs
Vesicles	Develop in twelve to twenty four hours are rarely umbilicated collapse on pricking all stages papules vesicles and flattened scabbing puckered pocks appear together	Papules hard and shotty are slow in developing vesico pustules are more commonly umbilicated eruption more uniform and the scabbing margin is not puckered

*From Measles* —The diagnosis from measles may be made as follows —

<i>Sign or Symptom</i>	<i>Measles</i>	<i>Smallpox</i>
Catarrhal symptoms	Lachrymation coryza cough present from the beginning and marked	Usually absent but there may be some conjunctival effusion
Filatow or Koplik's spots	Usually present	Absent
Eruption	Appears on the third to fourth day as minute pink papules behind the ears on the forehead chin cheeks neck limbs and chest papules not hard or shotty	Initial measly eruption on the first or second day on face trunk and limbs simultaneously If partial appears in the abdominal area Papules hard and shotty
Temperature	Reaches its height with the appearance of the rash	Falls with the appearance of the rash

*From Influenza etc* —In German measles the initial severe symptoms are absent and in the fourth disease the face is free while in influenza the typical eruption fails to appear

*From Typhus* —The diagnostic features are —

<i>Sign or Symptom</i>	<i>Typhus</i>	<i>Smallpox</i>
Erythema	Appears on the third to fourth day of the illness	Appears on the first or second day of the illness
Typical eruption	Petechial Appears on the fourth or fifth day and is rarely seen on the face	Papulo pustular Appears on the third or fourth day and is common on the face

*From Hansen's Diseases*—Any case of high fever of an acute nature associated with purpura may be smallpox. It is extremely difficult to separate

lymph an inspector of vaccination or more according to the needs of the country and a series of native vaccinators

## ALASTRUM

Alastrum is considered. One of the earliest descriptions by Anderson in

acute and virus

The question which has been much debated is whether it is smallpox, chickenpox, or a new disease halfway between the two. In the first place there are still some persons who do not believe in the difference between variola and varicella, but their objection is usually disregarded. In the second place alastrum differs from varicella because of—

1. Confluence of the vesicles in certain cases
2. Its frequency among adults
3. The partial protection by Jenner's vaccine



3 There is no secondary fever in children

4 Though Jenner's vaccine is in some degree protective the disease can occur after recent successful vaccination

5 Jenner's vaccination can be successfully carried out shortly after an attack of alastrim

6 According to some authorities an attack of smallpox does give a lasting immunity to alastrim

Provisionally we may conclude that it is probably a slightly different form of disease from true variola

**Symptomatology**—It begins with high fever, severe pains, and vomiting with very often delirium. The rash comes out on the third day when the



FIG 680—ALASTRIM (After Ribas)

It feels so well  
 fever usually  
 lay Papules  
 scarring but

recent  
 of contacts

## VACCINATION

o cur  
T

countries

The capillary tubes are filled in a vacuum in the Entrescan filling machine and should be tested for vaccine activity before being issued

Lanolated lymph not being sterile, is more apt to contain a large number of micro organisms. Dried lymph has been placed upon the market and

before being utilized for vaccination

The slight redness and swelling due to the scratches disappear in twenty four hours while the papules appear on the third to fourth day and are succeeded by the vesicles which become umbilicated pustules about the eighth to ninth day. These quickly begin to dry in the centre and form a scab which falls off about the fourteenth to twenty first day and leaves a scar.

### VACCINATION RASHES

Generalized Vaccinia

in  
Ar  
va

pustules and these to scabs

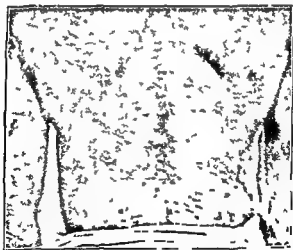


FIG 681—GENERALIZED VACCINIA

In a typical case the rash appears twelve to twenty two days after vaccination requires some six days to reach its full development, and another eight days to decline to the stage when the scabs are thrown off and the patient is convalescent.

no sign of septic infection in the disease of which the following is a typical case—

exceed 102° F

with vaccination

Localized gangrenous vaccinia on the other hand is quite different. It is not septic, but vaccinal in origin and it is not generalized but localized to the vaccination area.

Acland Crocker Balzer and Hutchinson have met with similar cases

the vaccination

Treatment At first the arm was to be made aseptically and covered with cotton-wool, loosely held in position by first a bandage and then a moist kerchief.

several hundreds

The blood showed no parasites but there was a leucocytosis and a marked  
 detachment of twenty four were vaccinated on February 10, 1914, and the

From *graw craw* (in the restricted sense of the word) by the absence of the horny consistency, and by the smaller size of the vaccinal papules  
 From *lichen convex* by being non follicular and by being an acute and not a chronic eruption.

to the lymph

**Treatment.**—The essential treatment is rest and quiet. On quinine by the mouth and an antiseptic ointment—*e. g.*, carbolic ointment—for the skin,

INFLUENZA

Remarks. In cases of influenza the temperature is usually raised to 101° to 103° F.

that the virus isolated in cases of influenza consists of minute Gram positive, roundish, coccus like bodies, varying from 0.15  $\mu$  to 0.5  $\mu$  capable of passing

1900	1901	1902	1903	1904	1905
------	------	------	------	------	------



130 652 --TEMPERATURE CHART OF INFLUENZA

attempt to impress upon the tropical practitioner the necessity of recognizing this complaint, which is very apt to be mistaken for pappataci and dengue

catarrhal symptoms appear the patient becomes convalescent and may recover completely or may fall into a relapse.

fever lasting however about a week.

In the *cerebral type* the onset may be sudden or gradual be with or without catarrhal symptoms and is associated with pains in various parts delirium aphasia hemiplegic or monoplegic symptoms and indeed may at times simulate a cerebral hemorrhage especially in afebrile cases. These are very fatal cases but at times recovery takes place often with permanent mental disturbance.

*Blood*—The blood practically shows no change. The total leucocytes vary from 8 000 12 000 while the differential count is within normal limits.

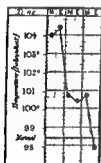


Fig 683

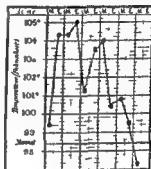


Fig 684

FIGS 683 AND 684—TEMPERATURE CHARTS OF INFLUENZA

**Complications and Sequelæ**—These are numerous and frequent but for

leucocytosis

In the tropics the most important points in the *differential diagnosis* are the separation of the malady from malaria dengue and pappataci fevers and from plague. From malaria it can be recognized by the absence of the

Treatment—It is important that the patient should remain in bed and be

with  
of the  
two

Prophylaxis—The patient should be isolated as far as possible and on every his room bedding clothing etc. should be disinfected. Vaccines have not been very successful

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## CHAPTER LIX

### WAR ZONE FEVERS

General remarks—Trench fever—*Icterus castrensis gravis*—*Icterus castrensis levis*—References

#### GENERAL REMARKS.

It may be thought that it is unnecessary to introduce the subject of the diseases of the different zones of the war into a work on tropical diseases, but many of the maladies which have affected the troops during the war in the Salonica area, in Egypt and

diseases, will be dealt with in the chapters which follow, but, excluding these, there are three conditions which merit a little consideration—viz, trench fever and the severe and mild forms

#### TRENCH FEVER.

*Lebris volhymica*

**Definition.**—A relapsing fever of as yet unknown origin, and spread by *Pediculus corporis* de Geer, 1778, commonly by the infected faeces being rubbed into the excoriated skin. It is characterized by a sudden

of five days' duration (more or less), and ending in complete recovery

**History**—As Strong has pointed out there is insufficient data to permit any of the diseases described by the ancients or in mediæval or modern times being connected with the disease

as seen in the British Army in which thousands of cases occurred between April and October. In January 1916 it was observed in Salonica by McGavin, White and Acland of No. 1 New Zealand Stationary Hospital. In May 1916 it was observed in Mesopotamia and in the same month was reported by Beauchant and Boudin as being present in the French Army in France and about this time Werner in Warsaw drew attention to its existence in the German armies and Hurst gave a good general account of the disease. In the same year McNee, Renshaw and Brunt showed that the disease could be transmitted to healthy men by intramuscular and intra-

possible source of infection in a certain hospital orderly who had never been in contact with patients and in whom the incubation was eighteen days from the first time he became infected with lice

They also proved that the louse was the infective agent and that the virus was naturally conveyed by its bite. This virus is present in the plasma, sometimes in the urine and occasionally in the sputum. Artificially the disease may be transmitted by rubbing lice feces, human infected urine or sputum into excoriated skin, but the incubation resembles that of the inoculation of infected plasma.

A little later in the same year the British Committee in London showed that lice bites did not produce the disease which however could be produced in healthy men by rubbing infected louse feces into excoriated cutaneous areas. Further they demonstrated that the incubation period was six to eight days and that blood taken from the infected men and injected into healthy men could reproduce the disease after an incubation of five days. Also in 1918 Couvy and Dujarric de la Riviere claimed that a spiro-

hemogregarina have been described as causal. In 1916 Toepfer found *Rickettsia* bodies similar to those described in 1909 by Ricketts in Rocky Mountain spotted fever and by

Ricketts and Wilder in 1910 in typhus. In 1917 Da Rocha Lima called the bodies found in trench fever *Rickettsia quintana* to distinguish them from those found in typhus (*R. prowazekii*) and those occasionally seen in normal lice (*I. pediculi*). These findings were supported in 1918 by Arkwright, Bacot and Duncan.

In 1919 Bradford Bashford and Wilson described minute bodies which they had succeeded in cultivating from the blood of patients using Noguchi's method of anaerobic cultures. These bodies are morphologically identical with *Rickettsia* bodies but they are Gram positive.

**Climatology**—The disease is known to exist in England, Flanders, France, Salonica, Greece, Macedonia, Tyrol, Galicia, Poland, Russia and Mesopotamia.

**Ætiology**—The ætiology has not been completely elucidated. Toepfer first found minute bodies in the intestinal contents of lice fed on trench fever patients which he considered to be *Rickettsia* bodies. These bodies were somewhat similar to those found in confirmed *Rickettsia* in Duncan's trench fever patients. They are minute Gram

negative organisms, round, oval or lancet shaped diplococci 0.3 microns in their shorter diameter by 0.3 to 0.4 in length. The first appearance in the excreta of lice being as a rule eight to ten days after the first infecting feed. They seem to be slightly smaller and less frequently lancet shaped than those found in typhus. The size of these bodies is such that they should not pass as a rule a bacterial filter but may occasionally pass a filter which retains

Gram positive.

The trench fever virus is considered to be a resistant filterative

the healthy by the agency of the clothes louse *Pediculus corporis* de Geer, 1778 and that it was usually conveyed by the bites.

The War Office Commission (composed of Byam, Carroll, Churchill, Dimond, Lloyd, Sorapure and Wilson) came to the conclusion after a series of important experiments that the infection was contaminative by means of the louse feces infecting scratches on the skin.

**Pathology and Morbid Anatomy**—Unknown.

**Symptomatology**—**Incubation**—Clinically the incubation period is believed to vary from fourteen to thirty days because this is

the time required to induce the disease experimentally by infected

During this stage there may be slight prodromata in the form of headache and pains in the limbs, but these may be absent.

*Attack*—The onset is sudden, the patient feeling giddy, weak,

and nystagmus may be present if the eyes are turned completely sideways

There are two curious types of this stage of the illness—viz, the *appetit*

The *Course* of the disease is that the next morning the temperature has fallen to normal or nearly normal, but it may remain high, the appetite is lost, the tongue is furred, and there may be pharyngitis and constipation. Now the patient complains of pains in the

seen on the chest, back, or abdomen. The spleen and more rarely the liver may now enlarge. The blood shows a leucocytosis, with a relative mononuclear increase. The red corpuscles are not reduced in amount, but the hæmoglobin is usually reduced, and

*Second Attack*—After the cessation of headache, pain about 101° F may cease, or the fever may recur in the evening, but usually ceases next day.

*Intermissions and Attacks*—These now succeed one another at regular intervals of about five days' duration, but the attacks of fever become shorter and shorter and the temperature lower and lower, so that unless care is taken to register the temperature every two hours about the time of an expected attack, the fever may be missed by the day and night temperature chart. Although

the patient does not feel or look ill, the pains and tenderness in the shins may become worse during each attack and may keep him from sleeping. The number of relapses is variable, and may reach six or seven.

*Rash*.—In a fairly large number of patients a delicate macular rash appears with each recurrence of the fever. It is generally seen on the chest and abdomen, and consists of small red macules

heart may occur

*Varieties*.—English observers recognize a short and a long type. In the former the fever lasts for three to four days, falls to normal, and after a few hours rises again for two to five days when it falls to normal and the fever stops. This variety resembles dengue fever.

tact fever, relapsing fever, malaria, smallpox, typhus and enteroidæ.

From *influenza* it may be distinguished by the absence of catarrhal symptoms and the mononucleosis.

From *dengue* and *pappataci* by the absence of leucopenia.

From *relapsing fever* by the absence of marked enlargement of the spleen and of the spirochætes in abundance in the blood.

From *smallpox* and *typhus* by the absence of the severe constitutional symptoms.

From *malaria* by the absence of the leucopenia and of the typical parasites from the blood.

From *enteroidæa* by the sudden onset and the pains in the muscles and bones, and by negative hæmoculture and serological reactions.

*Prognosis*.—This is good, as the mortality is nil and the recovery complete.

*Treatment*.—Pyramidon is strongly recommended, but constipation must be relieved, and the patient should be disinfected at once.

## ICTERUS CASTRENSIS GRAVIS.

**Definition.**—An acute specific fever caused by *Spiroschaudinnia* (1915) and of the spleen and a high

mortality

**History.**—The disease was first mentioned in Minorca in 1745 and then a described t was also nc number of mortality f probable that it was absent . In the present war it has occurred in the British, French, Italian Serbian and German armies, though it has been confused with the milder type. In 1886 Hirsch popularized the complaint and Weil again drew attention to the disease

1915 the Japanese investigators mentioned above discovered the causal agent and this has been confirmed by French, English, Italian, and German workers. Noguchi has found that strains of *S. icterohæmorrhagiae* isolated from patients in Japan and Belgium

in the kidneys and urine of wild rats in which it lives it is believed that the organism escaping in the rats urine and to a less extent in human urine, is the source of infection, which takes place through the skin when walking barefoot on sodden ground or by entering the alimentary canal in water

**Pathology.**—The jaundice is probably caused by obstruction to the smallest bile ducts brought about by a polymorphonuclear

infection

seven  
dental

onal shivering and high  
The patient is flushed  
and liver enlarge, and the  
superficial lymph glands may become palpable

**Course**—Jaundice appears two or three days after the onset  
The tongue is coated with a brown fur, and sordes form on the lips

and teeth. Vomiting may be present from the first and hiccough may also be troublesome. There may be pain and tenderness in almost every part of the body. The neck may be stiff and in these cases the cerebro-spinal fluid may be under pressure and contain an excess of polymorphonuclear cells and lymphocytes as well as albumen and bile.

The conjunctivæ may be injected and herpes may be present on the lips.

symptoms  
be

appear  
present between the fourth and ninth day. The pulse is slow in proportion to the temperature. The urine usually contains bile albumen granular and hyaline casts and sometimes a few red corpuscles and the spirochæte can be found after the first week.

Hæmorrhages from the lungs the stomach or more rarely the bowels may occur while epistaxis and purpura may be seen in severe cases.

**Termination**—The temperature drops by crisis or rapid lysis from the eighth to tenth day.

**Relapse**—There is often a return of the fever some few days after

the eighth and ninth day (it is difficult to see) and in the urine. Blood may be inoculated into the peritoneal cavity of guinea pigs in which illness supervenes after inoculation of not less than six days.

In order to distinguish the disease from other febrile illnesses

jaundice yellow fever and relapsing fever

From *camp jaundice* (*icterus castrensis lewis*) it can be diagnosed by the sudden onset the severity of the symptoms and the shortness of the illness.

From *enteric jaundice* which is rare it may be recognized by the presence of the polymorphonuclear leucocytosis and the absence



of leucopenia, and the usual signs of enteric fever, while blood cultures fail to demonstrate the typhoid bacillus and its allies

From *septic jaundice* it can be separated because the jaundice appears at an earlier date than in the latter disease, and the

type by the absence of the malarial parasites in numbers and by the presence of the slight leucocytosis

From *acute yellow atrophy* by the absence of the diminution of urea and uric acid, by the absence of leucine and tyrosin in the urine, and by the increase in size of the liver

From *typhus* by the absence of the peculiar facies, of the subcuticular mottling and the typical rash and by the presence of jaundice, which is only occasionally seen in that disease

From *blackwater fever* by the absence of hemoglobin from the urine.

From *pneumonia with jaundice* by the absence of right sided lobar pneumonia

From *yellow fever* by the pulse being rapid from the first and falling as the temperature falls

From *relapsing fever* by the absence of the malarial parasites from the blood  
 spirochetes of Weil's disease are found only in small numbers.

**Prognosis.**—This is serious the death rate being some 30 per cent

and Wani or of Martin and Pettit's immunized horse serum is recommended Salvarsan and its allies are useless Symptomatic treatment for constipation by salines and aperients is also to be remembered

**Prophylaxis.**—Disinfect the urine of patients for some nine weeks from the onset of the attack Catch and kill rats Disinfect the ground of the endemic area or remove the persons from this area

### ICTERUS CASTRENSIS LEVIS.

**Synonym**—Camp jaundice

**Definition.**—A slightly febrile disorder, characterized by mild febrile symptoms followed by a mild attack of jaundice lasting some two to eight weeks, after which there is a very prolonged convalescence

**Remarks.**—We have already noticed that older writers have

**Climatology**—It was common in Gallipoli the Balkans Italy and France

**Ætiology**—This is at present unknown. Certain authors consider it to be of paratyphoid origin but this is not so. Spirochaetes have been described in the urine by several observers including

complains of aches in various joints and muscles for weeks the skin

appeared others had remarked the staining of their shirts by

vomit. The temperature is normal or subnormal pulse usually

usual brownish colour at times there are periods of diarrhoea alternating with periods of constipation. The urine is scarce and

may feel very weak

**Diagnosis**—The characteristic features of the disease are—

organisms are destroyed by heat, and by the absence of the

symptoms

**Prognosis**—This is favourable *quoad vitam*, but the course of the disease may be prolonged, and for weeks and months the patient may be very depressed and weak.

**Treatment**.—There is no specific therapy, and hence treatment must be symptomatic, with rest in bed, milk diet, and urotropin in 10 grain doses three times a day.

**Prophylaxis**—The urine and faeces should be disinfected.

### Nanukayami.

A seven-day fever resembling a typical Weil's disease has been reported from several Japanese observers from the province of Fukuoka. Ido Ito and Wami have found that the causative agent is a spirochæte—*S. hebdomadis*—which is serologically distinct from *S. icterohæmorrhagica*. The normal host of the spirochæte seems to be the field mouse *Microtus montebelli*.

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## CHAPTER LX

# THE DIAGNOSIS OF A TROPICAL FEVER

Preliminary—Thermometric pseudo fever—Acute fevers—Fever of less than eight days' duration—Fever of more than eight days' duration—Chronic fevers—Summary

### PRELIMINARY.

The method of diagnosis is not the same in all cases, and the results are not the same in all cases.

We are endeavouring to place before him such information as we have found necessary to use in some twenty odd years of tropical life, and we may perhaps be pardoned if we mention some plain facts.

from the crown of his head to the soles of his feet, by ordinary clinical methods before any attempt is made to utilize the resources of the laboratory

A systematic clinical examination of every patient is most essential. It is the sum total of the various symptoms, none alone pathognomonic, which establishes the diagnosis in conjunction with which the results from the laboratory must be considered. A practitioner who is unable to come to some sort of a diagnosis without the aid of a laboratory should, in our opinion utilize his earliest spare moments in a course of post-graduate instruction

supervised by the practitioner, and not left to subordinates entirely,

otherwise mistakes will be possible. The specimens should always be collected and forwarded in the most aseptic method possible and should be accompanied by a statement recording the nature of the specimen, the date and time of its collection, the nature of the examination desired (which should not be vague—*eg.*, not 'urine for examination, but clearly stated, 'urine to be examined *quantitatively* for sugar'), and a brief statement of the salient features of the case and the suspected clinical diagnosis because it is the duty of the practitioner to help the laboratory in its work.

Finally, we desire most earnestly to impress upon the reader that the essential feature in the diagnosis of a tropical fever is a combination of clinical examination with laboratory work.

### THERMOMETRICAL PSEUDO-FEVER.

The practitioner working in high air temperatures should remember that the clinical thermometer, being of the maximum type, will rise quickly to some temperature corresponding to that of the air, and will remain thereat. Mistakes have been made, such as recording a number of cases of fever in an institution. In high air temperatures the thermometer should be taken out of cool water, placed in the patient's mouth; left there long enough to record the actual temperature, and then quickly replaced into cool water, in which it is examined.

The practitioner knows well the precautions *re* hot liquids or

### ACUTE FEVERS.

The acute fevers may, for purposes of diagnosis, be divided into those which have lasted less than eight days, and those which have been in progress eight or more days when seen by the practitioner.

#### FEVERS OF LESS THAN EIGHT DAYS' DURATION.

These fevers may be divided into —

- I Those exhibiting some striking physical sign
- II Those not exhibiting any striking physical sign

#### I. WITH SOME STRIKING PHYSICAL SIGN.

The physical signs to which we refer may be classified into —

- A Traumatism
- B Skin eruptions
- C Derangement of some bodily system
- D Localized derangement of some organ

## A TRAUMATISM

film if Laveran's parasites are present or marked mononucleosis it is probably malaria if polymorphonuclear leucocytosis is present it is probably septic fever

If the evidence is in favour of sepsis or if malaria has been excluded bacteriological examination of the blood and of the local discharge should be made and should include search for aerobic and if necessary anaerobic organisms

## B SKIN ERUPTIONS

These may be considered under the following headings —

- 1 Erythematous eruptions
- 2 Papular eruptions
- 3 Urticarial eruptions
- 4 Purpuric eruptions
- 5 Vesicular eruptions
- 6 Bullous eruptions.
- 7 Pustular eruptions
- 8 Pigmentation

## I ERYTHEMATOUS ERUPTIONS

## (A) ERYTHEMATOUS RASH GENERALIZED

I *The rash is more or less typical of scarlet fever —*

- (a) Onset of eruption less than forty eight hours after the appearance of the sore throat—*Scarlet fever*
- (b) Onset of eruption more than forty eight hours after the appearance of the sore throat—*Erythema or dermatitis scarlatiformis*
- (c) " " " "

ally quinine—

II *The rash has some maculo papular elements —*

- (a) Coryza and Koplik's spots have been or are still present—*Measles*
- (b) Coryza and Koplik's spots absent —  
With enlargement of the occipital cervical and other lymphatic glands—*German measles*



- 2 Œdema of face and eyelids and gastro intestinal disturbance eo inophthia and leucocytosis—*Trichinosis*
  - 3 No œdema of the face and eyelids Very rare—*Polymyositis*
  - 4 Swollen area affected with leprotic eruption Signs of leprosy in various parts of the body—*Leprotic fever*
- (b) Not associated with œdema —

- 1
- 2 There is a recent wound due to a rat bite or a history of a rat bite seven to twenty one days previously of which the wound may have healed Site of bite red and swollen becomes ulcerated Enlarged lymphatic glands Erythematous eruption with purple spots—*Rat bite fever*
- 3 History of a cat bite some ten to twenty one days before illness maculæ around site of bite and then on limbs infiltration of skin enlarged lymphatic glands Pains in muscles and joints Splenic enlargement Fever relapsing in type—*Cat bite fever*
- 4 Pains and aches all over the body but constitutional symptoms not very severe Rash appears with the fall of the temperature on the third day No malarial parasites in the blood *Stegomyia* (or *Culex*) mosquitoes abundant Endemicity of dengue-like fevers known—*Dengue*

## 2 PAPULAR ERUPTIONS

These eruptions often form part of the evanescent early symptoms of some fever and are therefore difficult to arrange in a satisfactory manner The practitioner will remember that drugs like iodides and the bromides may give rise to papular eruptions

- I *Catarrhal symptoms present* —  
Maculo papular eruption tending to form blotches Koplik spots present—*Measles*
- II *Catarrhal symptoms slight or absent* —  
(a) *Constitutional symptoms severe* —  
1 Markedly severe headache and backache Papules

Papules about the size of a pin's head on chest back and abdomen—*Dengue* (Van der Scheer's fever)



- 3 Pale dusky red papules or macules, fading into the normal skin, but slightly elevated, disappearing on pressure, at the margins of the axillæ, wrists, flanks, chest, back, shoulders, arms, and legs, with subcuticular mottling. Exclude malaria by blood examination—*Typhus fever*
- 4 Large red papules on face on the fifth to seventh day of illness, spreading over body as macules, after a visit to Akitaken and Nugataken, of the Island of Nippon, Japan. Enlarged lymphatic glands in some area of the body by lymphatic of bites by *mushi fever*

(b) Constitutional symptoms not severe and not following recent vaccination—

- 1 Rash of maculo-papules, circular, discrete not as bright or as elevated as measles. No Koplik spots. Occipital and other lymphatic glands enlarged. Pink eye present—*German measles*
- 2 Fever slight or, at times of epidemic, absent in some cases. Rash general composed of bright pinhead papules or red macules (*sic* morbilliform), associated with itching and with a few macules on the palms and soles—*Papular fever* (Castellani and Chalmers)

(c) Constitutional symptoms not severe, following recent vaccination—

- Rash composed of papules and papulo vesicles of large pinhead size appearing some seven to nine days after vaccination—*Vaccine lichen*

### 3 URTICARIAL ERUPTIONS

- (a) Fever slight, after ingestion of certain foods—*Febrile urticaria*
- (b) Worms present—*Helminthic febrile urticaria*

### 4 PURPURIC ERUPTIONS

Purpuric puncta may be caused by flea bites and pediculi, and

Bright's disease, and jaundice may be associated with purpuric rashes. In fevers as a rule it points to septicæmia, and is more of a prognostic than of a diagnostic value even in epidemic cerebro-spinal meningitis, in which, in our tropical experience, it is rare.

Peliosis rhe

joint, ■ v

no fever

Clear signs of the disease causing the purpuric eruption —

(a) Yellow tinge in skin or eyes —

1 Urine black from hæmoglobin—*Blackwater fever*

2 Urine without hæmoglobin —

(A) *Bile in the urine* —

(i) Examine blood and urine for the peculiar sprochæte with its central minute waves—*Icterus castrensis gravis* (*Weil's disease*)

(ii) Spirochetes of doubtful pathogenicity may be present or they may be absent, little or no fever—*Non febrile jaundice* (*camp jaundice*)

(B) *Bile not in urine* —

Severe fever examine blood for malarial parasites—*Malarial fever*

(C) *Albumen in urine* —

Epigastric tenderness. Faget's sign—*Yellow fever*

(b) Without yellow tinge in the skin or eyes —

1 Retraction of head Kernig's sign present Lumbar puncture reveals meningococci—*Epidemic cerebro spinal meningitis*

2 With developed disease—e.g. typhoid diphtheria scarlet fever smallpox measles (hæmorrhagic conditions known in the tropics)—*Septicæmia condition*

3 With buboes or marked pneumonic symptoms Examine blood culturally sputum and fluid from enlarged lymphatic glands microscopically for plague bacilli—*Plague*

4 Without marked signs of any disease Blood culture—*Septicæmia*

### 5 VESICULAR ERUPTIONS

T

I *Onset with severe constitutional disturbance* —

Rash on third to fourth day shotty papulæ becoming vesicles fifth to sixth day Vesicles circular tense umbilicated and multilocular—*Smallpox*

II *Onset with mild constitutional symptoms, but with severe local pain generally along a nerve but in any case confined to one region* —

D. 1. 1. 2. 3.

like or flattened become umbilicated. no inflammatory areola leave scars—*Vesicular fever* (Castellani and Chalmers)

III *Onset mild and without severe local pain without history of recent vaccination —*

Rash on first to third day. Often first sign of illness. Appears on back, chest and abdomen. First in form of pale red macules often with raised centre quickly developing into superficial unilocular vesicles some of which may become umbilicated—*Chicken pox*

IV *Onset mild history of vaccination twelve to twenty two days or more before eruption —*

Generalized vesicular eruption not umbilicated at first and preceded by a papular rash. Vesicles become umbilicated—*Generals ed vaccinia*

## 6 BULLOUS ERUPTIONS

Bullous eruptions may be caused by plants and drugs. In newly-born children bullæ on the hands and feet suggest congenital syphilis

- I There is a well defined raised erythematous area upon which the bullæ are present. Bullæ to be examined for streptococci—*Erysipelas*
- II No such area present but wounds enlarged lymphatic glands with severe constitutional symptoms common in the tropics. Flaming blood and bullæ for organisms—*Septic periphagus*
- III In recently born children examine bullæ for streptococci and other pyogenic organisms—*Pemphigus neonatorum*

## 7 PUSTULAR ERUPTIONS

It will be remembered that there are pustular syphilides unattended by fever and pustular tuberculides of which fever is not a marked sign

I *Clear history of recent vaccination with Jennerian vaccine —*

Small dark coloured or black centre in the vaccine area surrounded by dark reddish swollen area on which are the vaccinal vesicles and pustules. Around this a bluish area the whole surrounded by a wide erythematous blush—*Gangrenous vaccinia*

II *No history of recent vaccination —*  
 (a)

amine bacteriologically for *Bacillus anthracis*—*Malignant pustule*

- (b) Generalized pustular eruption in a patient who has been seriously ill for six days or more with at times swelling of eyelids lips or eyes—*Smallpox*

### 8 PIGMENTATION

In acute fevers the important cutaneous pigmentation is the

I *Fever and spleen one or both enlarged —*

- (a) Abdominal tenderness. Typical spirochetes in blood—

(b)

(A) Hæmoglobinuria—*Blackwater fever and its allies*

(B) No hæmoglobinuria severe symptoms albuminuria black vomit etc—*Yellow fever*

II *Liver and spleen not enlarged —*

- (a) Symptoms mild. Fever slight or absent. No signs or symptoms of pneumonia—*Icterus castrensis levis (camp jaundice)*

- (b) Physical signs and symptoms of pneumonia. Pneumococcus in sputum—*Pneumonia*

### C BODY SYSTEMS

Fevers associated with some marked sign or symptom directing attention to a given system of the body may be arranged according to the system deranged as follows —

- A Derangements of the Alimentary Canal
- B Derangements of the Respiratory System
- C Derangements of the Circulatory System
- D Derangements of the Urinary System
- E Derangements of the Generative System
- F Derangements of the Lymphatic System
- G Derangements of the Muscular System
- H Derangements of the Osseous System
- I Derangements of the Connective Tissue
- J Derangements of the Nervous System



## B Symptoms pointing to the respiratory system —

## I Nose —

## (a) Acute rhino pharyngitis —

Examine secretion microscopically and culturally —

- 1 Spirochetes—*Spirochaeta rhino pharyngitis*
- 2 *Micrococcus catarrhalis* and similar organisms  
—Common cold
- 3 Influenza bacillus or filterable virus present—  
*Influenza*
- 4 Influenza bacillus or filterable virus with  
Streptococci—*Streptococcal complications of influenza*

## (b) Nose partially blocked —

Examine swabs microscopically and culturally for the  
Klebs Loeffler bacillus—*Diphtheria*

## (c) Larynx —

More or less stridor Examine swabs of throat for  
Klebs Loeffler bacillus—*Diphtheria*

## (d) Bronchi —

Signs of bronchitis with or without blood in the sputum  
Examine fresh sputum microscopically and if  
necessary by the dark ground illumination —

- 1 Eggs—*Paragonimiasis*
- 2 Spirochetes—*Bronchospirochaetosis*
- 3 Fungi—*Bronchomycosis*
- 4 Acid fast organisms —  
(A) Tubercle bacilli—*Tuberculosis*  
(B) *Nocardia*—*Pulmonary Nocardiasis*

## (e) Lungs and pleura —

Physical signs of inflammation of the lungs or pleura  
or both —

- 1 Expectoration chocolate coloured—*Lister abscess*
- 2 Expectoration bloody or rusty Examine  
microscopically for the same points as under  
bronchi and for the pneumococcus—*Pneumonia*
- 3 Expectoration not chocolate-coloured nor  
bloody—*Diseases of lungs and pleura other than above*

## C Symptoms pointing to the circulatory system —

- 1 Marked collapse after exposure to great heat (especially associated with high atmospheric humidity) or to the sun's rays. There may or may not have been initial fever—*Heat syncope*
- 2 Disturbed action of the heart with severe constitutional symptoms and petechial eruption—*Infective endocarditis*

## D Symptoms pointing to the urinary system —

## (a) Urine black, due to hæmoglobin —

- 1 Following on a dose of quinine—*Quinine hæmoglobinuria*
- 2 With malarial parasites in numbers in blood—*Malarial hæmoglobinuria*
- 3 Not associated with quinine or malarial parasites in numbers in the blood—*Blackwater fever*
- 4 Associated with the administration of some drug such as chlorate of potash for a sore throat—*Toxic hæmoglobinuria*

## (b) "

BLACK VOMIT etc —*Blackwater fever* or *Yellow fever*

## (c) Passage of large quantities of urine —

Examine for malarial parasites or other signs of malaria — e.g. enlarged and tender spleen—*Pernicious malaria*

## (d) Bile in the urine —

- 1 Associated with hæmoglobin—*Blackwater fever*
- 2 Not associated with hæmoglobin —

## (A) In epidemic form —

Examine blood and urine for spirochætes

- (i) Mild cases—*Icterus castrensis levis*
- (ii) Severe case with hæmorrhages—*Icterus castrensis gravis* (*Weil's disease*)

## (B) Not in epidemic form —

No spirochætes During or after an attack of typhoid or paratyphoid fever—*Bacillary jaundice* (*Enteric jaundice*)

## E Symptoms pointing to the reproductive system —

## (a) Chill sudden pain and swelling along the spermatic cord with often severe fever but no erysipelatous

- (b) " " " " " "  
*funiculitis*  
 the aseptically  
 streptococci and

## F Symptoms pointing to the lymphatic system —

## (a) Cervical glands enlarged —

- 1 Most marked on the left side associated with obstinate constipation and mild symptoms  
 Puncture of glands reveals no organisms—*Pfeiffer's glandular fever*

- 2 Most marked in the posterior triangles of both sides of the neck History of residence in sleeping sickness areas Glandular fluid obtained by puncture shows trypanosomes—*Trypanosomiasis*
3. Enlarged glands in neck and other parts Œdema of face with characteristic crepitation Fugitive œdemas in various parts Enlarged and tender spleen Enlarged liver Increase in size of the thyroid gland Residence in Tropical South America—*Chagas disease*

(b) *Lymph glands anywhere enlarged* —

- 4 Pain in some lymph glands tender enlarged freely movable under skin Search area drained by lymphatics going to gland for circular vesicle or small black or brownish necrotic area indicative of a bite Puncture of glands shows no bipolar plague bacilli History of residence in the Akitaken and Nugataken of the Island of Nippon Japan—*Tsutsugamushi disease*

(c) *Inguinal or axillary glands enlarged* —

- 5 Acute onset high fever great prostration Puncture of glands reveals plague bacilli—*Plague*
- 6 Gradual onset slight fever Very mild symptoms malaise pain on walking Inguinal or crural glands enlarged hard very painful on pressure Puncture shows sterile fluid—*Climatic bubo*
- 7 Glands enlarged inflamed or suppurating with chancre on penis septic wound or ulcer or gonorrhœal infection—*Septic infections*
- 8 Occurring in the course of one of the enteric fevers—*Intestinal infections*
- 9 High fever lymphangitis associated with an erysipelatous condition of the skin Blood examination during night (or during the day in certain cases) reveals microfilaria—*Filarial lymphadenitis*

G *Symptoms pointing to the muscular system* —

- (a) Remittent or intermittent fever with rheumatoid pains and abscesses in various parts of the body—*Myositis purulenta tropica*
- (b) Remittent fever with rheumatoid pains but no abscess formation Œdematous patches often present marked eosinophilia—*Trichinosis*

H *Symptoms pointing to the osseous system* —

- (a) Pain and tenderness especially near a joint—*Osteomyelitis*



- (b) Sudden attack of fever, with great tenderness over, and pain in, the os calcis or other tarsal bone, which begins to increase in size—*Endemic enlargement of the os calcis*

*Symptoms pointing to the connective tissue:—*

Rigors with fever and aching or dragging sensation, and outline of a worm under the skin of affected area—*Dracontiasis*

*Symptoms pointing to the nervous system —*

- (a) Almost any acute sign or symptom pointing to the nervous system, including signs of mania, melancholia, or dementia and associated with fever, with or without enlargement of the spleen. Examine blood for malarial parasites or mononucleosis—

- *Malaria*

- (b) Signs of meningitis present—*e.g.*, Kernig's sign, retraction of the head etc. Examine cerebro-spinal fluid —

1 Polymorphonuclear leucocytes and cocci present—*Epidemic cerebro spinal meningitis*

2 Trypanosomes present, also in juice from enlarged neck glands. Residence in Tropical Africa—*Sleeping sickness*

- (c) Signs of acute alcoholism —

If picked up by the police, even if there is a smell of alcohol examine spleen and take blood films if necessary. Drunk or dying in the tropics is often a question of alcoholism or malaria. Fever may be absent in both instances—*Acute alcoholism or malaria*

#### D. DERANGEMENT OF SOME ORGAN

The signs and symptoms associated with some organ of the body may be considered under the following headings —

- 1 The Spleen
- 2 The Liver
- 3 The Pancreas
- 4 The Suprarenal Capsules
- 5 The Parotid

*The spleen —*

- 1 *Enlargement slight —*

Rose-coloured spots on the abdomen. Symptoms of typhoid fever. Make blood cultures and faecal cultures—*Enteroida fevers*

II *Enlarged and tender* —

- (a) Examine blood films for malarial parasites and for spirochaetes—*Malaria* or *Relapsing fevers*
- (b) With œdema of the face and enlargement of the thyroid and lymphatic glands and liver Residence in South America—*Chagas American trypanosomiasis*

III *Enlargement considerable* —

- (a) Generally a history of illness lasting some time of which present fever is only a recurrence Firm enlargement Malarial parasites in blood—*Exacerbation of chronic malaria*
- (b) No malarial parasites in the blood —  
Great increase in white blood cells with myelocytes—*Leukæmia*
- (c) No malarial parasites and no great increase of leucocytes in the blood —
  - 1 Splenic or hepatic puncture shows Leishman Donovan bodies—*Kala azar*
  - 2 Shows no Leishman Donovan bodies — *Fœbril splenomegaly*
  - 3 Toxoplasma bodies present—*Toxoplasmosis*

B *The liver* —

*Enlarged and tender* —

- 1 Pain in the right shoulder rigidity of right rectus diminution of movement of right side of the diaphragm Examine motions for amœbic cysts and the blood for mononucleosis (present) and malarial parasites (absent)—*Amœbic liver abscess*
- 2 Signs of severe septic infection jaundice etc II

3

C *The pancreas* —

- I With intense pain in the upper and left part of the abdomen which is distended with gas vomiting and constipation—*Acute pancreatitis*
- II Signs and symptoms of diabetes threatened Kussmaul's coma Recurrent attacks of fever every other day Examine for malarial parasites if absent and if only polymorphonucleosis give a few doses of quinine and note action on fever—*Malaria and diabetes*

D *Suprarenal capsules* —

Signs suggestive of acute peritonitis—: *e* high fever distended tympanitic abdomen quick pulse No effusion into abdominal cavity Examine blood for malarial parasites and for mononucleosis If absent give quinine and again test blood  
—*Acute malaria attacking suprarenals*

E *Parotid glands* —

Painful tender swelling of parotid especially if bilateral—  
*Mumps*

## II. ACUTE FEVERS WITHOUT STRIKING PHYSICAL SIGN.

A *Patient is carrying on his ordinary work* —

I Fever is intermittent every third or fourth day Examine spleen for enlargement and tenderness and examine blood for malarial parasites and mononucleosis which may be absent Clinical symptoms alone may be positive—  
*Malarial fevers*

II Fever is quotidian Examine spleen for tenderness and enlargement Examine blood for malarial parasites and mononucleosis If none give quinine and note action on fever—*Malaria*

III No malarial parasites and quinine therapy without effect —

(a) Pulse dicrotic slow in proportion to the temperature History of several days indisposition Tongue furred constipation or diarrhoea Gurgling on pressure in right iliac region Make blood and

(b) P<sub>1</sub> ture Attack sudden with at first pain and tenderness which later disappear in the region of the appendix No malarial parasites in blood—*Gangrenous appendicitis*

(c) Abrupt onset catarrhal symptoms with sensation of considerable illness and with generalized pains often in epidemic form—*Influenza*

(d) With or without signs of bronchitis enlargement of  
 l . . . . .  
 v . . . . .

or tuberculosis

(e) G very night  
 and limbs  
 ' *melitensis*

B *Patient unable to carry on his usual duties* —

- (a) Liver and lymphatic glands enlarged Spleen not enlarged Examine blood Signs of great destruction of red blood corpuscles (presence of *Bartonella bacilliformis*) Residence in Peru—*Oroya fever*
- (b) Sudden onset with injected conjunctivæ (pink eye) high fever comparatively slow pulse Severe rheumatoid pains Liver and spleen normal Patient irritable with pain in head and eyes and may be delirious Endemic area for *Phlebotom* is flies—*Pappataci fever*
- (c) Sudden onset with severe pain in some part of the body or all over the body With or without enlargement of the lymph glands with generally a maculo papular eruption on the third or fourth day Conjunctivæ injected Fauces congested Pulse increases proportionately with the fever Endemic area for stegomyia (perhaps also for *Culex fatigans*)—*Dengue fever*
- (d) Sudden onset Hyperæsthesia over shins Pains in the legs Often slight splenic enlargement Mononucleosis in blood May or may not be history of association with lice Blood examination excludes malaria relapsing fever etc—*Trench fever*
- (e) Sudden onset with or without rigors and pains Examine blood for malarial parasites—*Malaria*
- (f) Gradual onset Signs of enteric fever Mal e blood and fecal cultures—*Enteroides group of fevers*
- (g) Blood examination reveals marked polymorphonuclear increase Examine gums teeth ear nose throat fingers toes bones and every orifice of the body for possible source of infection make blood cultures—*Septicæmia*
- (h) Blood examination Examine night and day blood for microfilaræ—*Elephantiasis*
- (i) Examine fæces for intestinal eggs especially after a purgative—*Toxæmia due to intestinal worms*
- (j) Gradual onset with marked pains in the joints profuse sweating high fever and relatively slow pulse Furred tongue—*Undulant fever*
- (k) Sudden onset with hyperpyrexia delirium or coma associated with high atmospheric temperatures—*Thermic fever (heat stroke)*
- (l) Sudden onset with or without history of fever Syncope associated with high atmospheric temperatures—*Heat syncope*

## FEVERS OF MORE THAN EIGHT DAYS DURATION

Fevers of more than *eight days* and less than *six weeks* duration may be classified as follows —

### A *Fever of intermittent type* —

With malarial parasites or pigment in blood or with enlarged spleen —

I Fever every day—*Quotidian malaria*

II Intermittent fever every third day—*Tertian malaria*

III Intermittent fever every fourth day—*Quartan malaria*

### B *Fever of the relapsing type* —

I Without malarial parasites or pigment and not reacting to quinine therapy. Intervals between attacks several days. During attack spirochetes in blood—*Relapsing fevers*

II With malarial parasites and no signs of spirochætes and reacting to quinine therapy—*Malaria*

III Without parasites and only one or two relapses not reacting to quinine therapy. After a long fever presumed or proved to be enteroidæa in type. Examine fæces and urine for enteroidæa organisms—*Enteroidæa type of fever*

### C *Fever remittent or continuous* —

I Reacting to quinine therapy—*Malaria*

II Not reacting to quinine therapy

## A WITH MARKED PHYSICAL SIGNS

### 1 *Well defined local pain and tenderness* —

Examine blood films. Leucocytosis blood cultures urine cultures. Lastly examine cerebro spinal fluid (earlier if head or spine symptoms)—*Septicæmiæ or toxæmiæ due to foci of deep suppuration*

### 2 *Signs of lung disease* —

Examine sputum —

(a) Tubercle bacilli—*Tuberculosis*

(b) Other organisms and signs of pneumonia—*Broncho pneumonia*

### 3 *Organic cardiac murmurs* —

With or without petechial eruptions. Signs of gonorrhœa or rheumatism—*Infective endocarditis*

### 4 *Nervous symptoms* —

Pain in the head retraction of the head Kernig's sign. Examine cerebro spinal fluid—*Meningitis*

5 *Skin eruptions* —(a) Rose red spots—*Enteric fevers*(b) Flushing of the face with subcuticular mottling and severe symptoms. Typical eruption on fourth day—*Typhus fever*(c) Purulent discharge from nose. Bullæ nodules and ulcers in skin with papulo-pustular eruption. Work with horses—*Glanders*(d) Pustular eruption—*Glanders*(e) Dark or black pigmentation—*Addison's disease*6 Enlarged lymphatic glands—*Hodgkin's disease*7 Tenderness in a bone especially near a joint. Blood cultures—*Osteomyelitis*

## 8 Nodules and tenderness in muscles. Puncture the nodules and examine —

(a) Pus—*Purulent myositis*(b) Filaria—*Filariasis*9 *Splenic enlargement* —

Examine blood —

(a) Marked increase of lymphocytes or leucocytes with myelocytes—*Leukæmia*(b) Malarial parasites or pigment in leucocytes—*Malaria**Splenic or hepatic puncture* —(a) Malarial parasites or pigment—*Malaria*(b) Leishmania parasites—*Kala azar*(c) Absence of Leishmania parasites—*Splenomegaly febrile form*(d) Toxoplasma bodies present—*Toxoplasmosis*

## B WITHOUT MARKED PHYSICAL SIGNS

A *Intermittent fevers* —I Fever every third or fourth day—*Malaria*

II Fever every day. Examine blood —

1 Malarial parasites or distinct mononucleosis—*Malaria*2 Malarial parasites absent. distinct polymorpholeucocytosis—*Septic fevers*B *Relapsing fevers* —Fever for several days after period of apyrexia—*Relapsing fevers*C *Remittent and continuous fevers* —I Benefited by quinine with or without parasites in blood—*Malaria*

## II Not benefited by quinine —

- (a) Test the serum reactions for typhoid and the paratyphoids and the other common enteroid organisms of the period and locality. Confirm by blood cultures—large quantities 10 c.c. of blood taken at night—or by fecal cultures—*Enteroides group*
- (b) Test for Mediterranean fever by serum reactions and blood cultures—*Undulant fever*
- (c) Culture of aseptically collected urine—*Pyelitis*
- (d) Examine motions for eggs of intestinal worms—*Intestinal toxæmias due to worms*
- (e) Wassermann reaction—*Syphilis*
- (f) Cuti reaction for tuberculosis—*Tuberculosis*
- (g) Other causes having been excluded. Bodily temperature  $99^{\circ}$ – $101^{\circ}$  F shows only slight rise once a day. Patient indisposed during the attack—*Low intermittent fever*
- (h) Patient residing in locality with high atmospheric temperatures. Patient not indisposed during the attack—*Low heat fever*
- (i) Same as in (g) but in children with higher temperatures  $103^{\circ}$ – $104^{\circ}$  F or more—*High intermittent fevers*

## CHRONIC FEVERS

By the term chronic fevers we mean those which continue longer than six weeks

*Intermittent in type* —

II Occurring every fourth day with enlargement of the spleen and malarial parasites or pigment in the blood or yielding to quinine therapy—*Quartan malaria*

III Occurring every day with enlargement of the spleen and malarial parasites yielding to quinine therapy—*Quotidian malaria*

*Relapsing in type* —

Attacks of fever lasting a few days separated by intervals of several days with severe symptoms. Examine blood for spirochaetes. If necessary inject monkeys and examine blood during an attack of fever for spirochaetes—*Relapsing fevers*

C *Remittent or continuous in type* —I Benefited by quinine therapy—*Malaria*

II Apparently not benefited by quinine therapy —

(a) Ulcers or tumours present in some part of the body  
Examine thoroughly including nose naso-pharynx,  
and all apertures of body Especially examine the  
teeth particularly crowned teeth or bridges Ex-  
amine fingers and toes carefully—*Septic infection or  
absorption*

(b) *Cutaneous dark pigmentation a marked feature* —

1 Examine spleen for enlargement and blood for  
malarial parasites or mononucleosis Insuffi-  
cient quinine administered—*Malaria*

2 Fever generally absent No signs of malaria  
V miting at times Weakness etc—*Addison's  
disease*

(c) *Splenic enlargement a marked feature* —

I Examine blood films —

No malarial parasites seen

(A) Marked increase in white cells lymphocytes  
or with myelocytes—*Leukæmia*

(B) Having excluded leukæmia but not before  
examine blood obtained by splenic punc-  
ture —

1 Malarial pigment or parasites present—  
*Malaria*

2 Leishman Donovan bodies present—*Kala  
azar*

3 Leishman Donovan bodies absent—  
*Febrile splenomegaly*

4 Toxoplasma like bodies present—*Toxo-  
plasmatic febrile splenomegaly*

5 All parasites absent—*Pseudo kala azar*

(d) *Œtéma a marked feature* —

I Examine blood for malarial pigment parasites  
or mononucleosis and the spleen for enlarge-  
ment—*Chronic malaria*

II No signs of malaria —

(A) Examine motions for eggs of intestinal  
worms especially ancylostoma ova—  
*Ankylostomiasis*

(B) No eggs or signs of worms In South  
America. Examine blood during an attack  
of fever for trypanosomes—*Chagas  
disease*



(e) *Intestinal indigestion a marked feature* —

- I Examine motions after test-meal for muscle fibres etc. Examine urine or not a marked

- II Attacks of fever a marked symptom No muscle fibres etc., in motions No Cambridge's urinary reaction Examine faeces after a purge for eggs of intestinal worms, and if absent, for micro organisms of proteus and allied groups—  
*Intestinal infections and toxæmias in helminthiasis*

(f) *Enlarged lymphatic glands a marked feature* —

- I Fever not a marked feature, glands very much enlarged in many parts of the body. No very great increase in the number of leucocytes—  
*Hodgkin's disease*
- II Attacks of fever a marked feature Glands only moderately enlarged especially in the posterior triangles of the neck Residence in Tropical Africa Examine gland juice for trypanosomes—  
*Sleeping sickness*

## Summary.

This small sketch of the diagnosis of certain tropical fevers may be found useful when read in conjunction with the preceding chapters. We would, however, again emphasize the point that the only method of diagnosing fevers is by long bedside experience, associated with careful laboratory work.

**SECTION B**  
**GENERAL DISEASES**

**DIVISION I CAUSATION ANIMAL PARASITES**

**SUBDIVISION A DUE TO PARASITIC PROTOZOA**

Framboesia Tropica  
Verruga Peruviaua  
Rhinosporidiosis and Sarcosporidiosis

**SUBDIVISION B DUE TO PARASITIC WORMS**

Paragonimiasis  
Katayama Disease  
Filariasis

**SUBDIVISION C DUE TO PARASITIC ARTHROPODS**

Myiasis  
Porocephalosis

**DIVISION II CAUSATION VEGETAL PARASITES**

Leprosy  
Histoplasmosis

**DIVISION III CAUSATION CHEMICAL**

**SUBDIVISION A DUE TO CHEMICAL DEFICIENCY**

Beri Beri and Epidemic Dropsy

**SUBDIVISION B DUE TO POISONS**

Tropical Poisonings

**DIVISION IV CAUSATION UNKNOWN**

Pellagra



## CHAPTER LXI

# FRAMBOESIA TROPICA

Synonyms—Definition—History—Geographical distribution—Ætiology—  
 Histopathology—Symptomatology—Diagnosis—Prognosis—Treatment  
 —Prophylaxis—References

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and other  
German  
which was  
berry like  
suggested  
and Iman  
atta Cas

tellans treponemosis and Violle the term cutaneous spirochæ  
 tosis Other local names are gattoo (West Coast of Africa)  
 dubi (Gold Coast) framosi (Calabar) aboukine (Gaboon)  
 nkoulou tetra (Congo Coast) momba (Angola) parangi  
 (Ceylon) buena (Burma) puru (Borneo Federated Malay  
 States) patek (Dutch Indies) tonga (New Caledonia and  
 Loyalty Islands) coco (Fiji) tona (Tonga Island) lupani  
 tono (Samoa) galis pateros (some parts of the Philippine  
 Islands) hi mo (French Indo China)

usually translated Ali Abbas and Avicenna who wrote at the  
 end of the tenth century mention a disease called safat or  
 sahafati with symptoms not unlike those of framboesia but most  
 authors are of the opinion that the disease referred to by the two  
 Arabian physicians was syphilis The study of the disease first  
 began to engage the attention of European physicians after the  
 discovery of America Oviedo y Valdez (1478-1557) describes it in  
 his report (1648)  
 Roche-  
 report it  
 nong the

is, in 1718  
West Indies,  
the East,

where it was known by the name of 'anboyna pox,' or 'pimple'  
In the days of the slave trade, outbreaks of frambœsia frequently

Indies. Occasionally in the countries in which it is endemic the disease may increase to such an extent as to cause veritable epidemics. An example of such an epidemic occurred in Dominica in 1871, when two special segregation hospitals had to be built for frambœsia patients.

In 1769 an outbreak of a peculiar disease occurred in Scotland. It was called 'sibbens' or 'sivvens' (*sivvi* Celtic for raspberry), and was apparently imported by sailors belonging to a vessel coming from the West Indies which was wrecked off Wigton in Cumberland.

The so called 'button scurvy' of Ireland endemic there in the eighteenth and the beginning of the nineteenth centuries, the 'radesyge' which broke out in Sweden and Norway in 1710, and the 'mal de chicot' in Canada have likewise been considered by some writers to be forms of frambœsia.

allied conditions. The term 'boubas,' for instance is used by the inhabitants of Brazil for various ulcerative conditions, such as frambœsia, leishmaniasis, and blastomycosis but most of the medical South American authorities use it as a synonym for frambœsia. Unfortunately Breda used it to denote a form of leishmaniasis and caused much confusion. The experimental researches of one of us in cases of frambœsia contracted in different parts of the world (tropical America, East and West Africa, etc.) show that 'yaws,' 'pian,' 'boubas' and 'parangi' are merely synonyms, but it is possible that there may be several varieties of the spiro-

observers

proved by actual experiment that syphilis and al investigation of alue. His report,

published in 1891, has become classical

Among the more recent observers who have investigated the

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framboesia has been reported from Greece by several writers

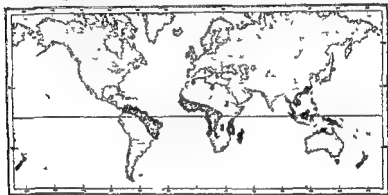


FIG 685—DISTRIBUTION OF FRAMBESIA TROPICA

*Africa*—The disease till recently was said to be very rare in the northern regions of the continent, though some cases were reported from Algeria the researches of Gabbi and Sabelli have demonstrated however, that it is common in Tripoli. It is apparently rare in Egypt, though according to some writers it is observed fairly frequently in the Sudan. It is very common on the West Coast, especially on the Gaboon River, in the Congo Free State, and in Angola. The disease is quite common in Nigeria

Government Hospitals during the last ten years has been on the average 3 500 per year, and it must be noted that the patients treated in hospitals represent only a small portion of all the cases

In India it is very rare though small outbreaks of the disease have been described by various observers. It occurs in certain parts of China but is unknown in Japan and the central and western regions of the Asiatic continent. It is present in the Philippine Islands.

*America*—It is very common in the West Indies and occurs in British Guiana, Venezuela, Colombia and Brazil. Cases have been reported from the southern United States but never from the northern States nor from Canada. Recently Wood has recorded a case in a white child in North Carolina.

*Australasia*—The disease is present in Northern Australia and occurs frequently in many of the Pacific Ocean islands—Samoa, New Hebrides, New Caledonia and Fiji. It is absent in New Zealand and Tasmania.

*Ætiology*—Different kinds of bacteria have been described as causative agents of frambæsia. Eijkman found some peculiar

disease. In February 1905 Castellani observed a *Treponema* or spirillum as he thought it at the time. This organism which he called *T. pertenue* is now generally admitted to be the cause of the disease. For the description of the organism see p. 457.

*Incidence of the T. pertenue in Frambæsia Lesions*—The presence of the *Treponema* is constant in the primary lesion and in the unbroken papules of the general eruption. It may be found in the spleen, lymphatic glands and bone-marrow. In the blood it has not yet been demonstrated microscopically though there is no doubt that the blood of the general circulation is infectious, inasmuch as monkeys inoculated with it develop typical yaws lesions in which the *Treponema* is abundantly present. The *Treponema* is absent in the cerebro-spinal fluid and generally in the tertiary lesions.

cases

*Inoculation Experiments of Frambæsia in Man*—Paulet in 1848 inoculated fourteen negroes with the secretion taken from frambætic granulomata. All of them developed frambæsia the inoculation period varying from twelve to twenty days when at the seat of inoculation in ten cases the first nodule appeared soon

yaws patients are not immune against syphilis is proved also by Powell and Nichols and others who have described several cases of syphilis supervening on yaws. Syphilitic patients may contract frambœsia naturally and experimentally.

*Inoculation Experiments in Monkeys and Other Animals*—Neisser Prowazek Halberstadter in Java and shortly afterwards Castellani in Ceylon have shown that monkeys are susceptible to frambœsia. According to their experiments the inoculation period varies from a minimum of sixteen days to a maximum of ninety-two. The appearance of the lesions developing at the seat of inoculation is practically the same in all cases—viz an infiltrated spot slowly increasing in size and soon becoming moist



FIG 686—MONKEY INOCULATED WITH FRAMBŒSIA

the secretion drying into a thick crust

bœsia can reproduce the disease in monkeys. The inoculation of the blood of the general circulation also may occasionally produce the disease. The inoculation of cerebro spinal fluid into normal monkeys has always proved negative.

Neisser Halberstadter von Prowazek in Java and later Castellani



in Ceylon, have proved that monkeys successfully inoculated with frambæsia do not thereby become immune to syphilis, and, vice

for frambæsia. According to Ashburn and Craig, monkeys of the species *Cynomolens philippinensis* are susceptible to frambæsia, but not to syphilis.

The following facts are in favour of the *T. pertenue* being the specific cause of frambæsia —

x In the non ulcerated papules, in the spleen, in the lymphatic glands of frambæsia patients, as well as in inoculated monkeys, the *T. pertenue* is the only organism present. No other germ can be demonstrated either microscopically or by cultural methods.

**PREDISPOSING CAUSES** — As is the case in other infectious diseases, dirt and other insanitary conditions favour to a certain extent the development and dissemination of the disease. The malady

is more common among the poorer class natives, while it is very rare among the better class natives, who live in cleaner, less overcrowded huts. Sex does not exercise any influence, nor does age to any great extent, though the disease is more frequently met with in children and young people. The native practitioners of Ceylon are inclined to ascribe an important predisposing influence to certain foods. Some incriminate a kind of fish called 'balla mai', others a cereal known as 'kurrakan'. In the West Indies the abuse of mango fruit

epithelial layers show many patches, in which the epithelial cells are swollen, vacuolated, and degenerating. Small, sharply circumscribed areas are also seen containing polymorphonuclear leucocytes and detritus. The layers near the corium and its processes are, in places, highly cellular, the connective-tissue being composed of elongated, papillary processes, the tips of which often nearly reach the surface. The corium is the seat of marked œdema. There is an diffuse cellular infiltration made up of polymorphonuclear leuco-

cytes large and small mononuclear leucocytes eosinophiles plasma cells mast cells connective tissue cells and some extravasated erythrocytes In the older nodules the plasma cells are present in such enormous numbers as to dominate all the others MacLeod has shown that there is no perivascular mononuclear infiltration so characteristic of syphilis nor any endothelial proliferation in the vessel walls The frambœtic lesion also differs from that of syphilis in affecting the epithelium rather than the cutis in the more considerable œdema and in the absence as a rule of the giant cells

When the frambœtic granulomata have reached a certain stage a very well marked hyperkeratosis is noticeable One of us has called attention to the appearance of the films taken in the usual way from the granulomata and stained according to Leishman's method In such films it is interesting to note the presence of a large number of polychromatic red blood cells of very different sizes some much larger than the normal erythrocytes some much smaller They are stained deep or light blue instead of pink and sometimes have a granular appearance The leucocytes present in the films frequently contain in their protoplasm and sometimes in their nuclei roundish or oval more or less deeply blue stained bodies which are probably polychromatic micro erythrocytes engulfed by phagocytes Some of these bodies present peculiar chromatin dots In such films the Treponemata are almost constantly found The Treponemata may be put in evidence also in

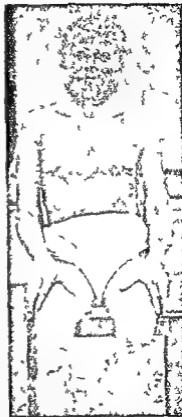


FIG. 687.—FRAMBŒSIA PRIMARY STAGE SHOWING THE PRIMARY LESION OR FRAMBŒSOMA BELOW THE RIGHT ANKLE

sections by using the Volpino

f the disease develop—deep ulcerations and gummatous-like nodules. A fourth period may perhaps be added (paraframbæscial affections). This division into three or four periods is of course somewhat arbitrary as symptoms considered to be characteristic of one period may make their appearance in another tertiary symptoms for instance may appear during the secondary stage.

It has been stated again and again that the whole course of the disease lasts from three to six months in children and six to twelve in adults but according to our experience it has a much longer duration and unless it becomes extinct after the secondary stage may extend to many years. Indeed we believe that in a certain number of cases although there are periods during which the patient is apparently free from symptoms the infection is merely latent and sooner or later gives rise to renewed manifestations.

**The Primary Stage** *Frambæsonia*—After a period of incubation varying in time between two to four weeks characterized often by signs of malaise rheumatoid pains headache irregular

ing a yellowish secretion which dries into a crust. Often at the place of inoculation several papules appear become moist and coalesce into a single element covered by a thick crust. If after some days the crust is removed the primary sore will appear as an ulcer not rarely of large dimensions with clean cut edges and a granulating fundus. This ulcer may heal leaving a whitish scar which may later become pigmented or in other cases it may develop into a nodular ulcer. In the tropics it is frequently called the "mother sore" or "mother ulcer".

In Columbia of South America Occasionally round it before the general eruption begins several smaller granulomata develop like satellites. The primary sore never feels indurated and is often painful during the first stage of development. Later it may be quite painless. Occasionally there may be pruritus. The proximal lymphatic glands may become hard and enlarged but they do not suppurate.

When the primary sore on one of the mammae develops on some crack or abrasion of the nipple and areola. In several other women the primary lesion was found on the skin of the trunk just above the hip this being due to the custom of the Ceylon woman carrying

her child astride of the hip. Any frambætic element present on the scrotum or nates of the child being continually rubbed against the skin of the mother, is likely to cause infection in the latter through any slight abrasion already present, or brought about by the friction. In men and children the primary lesion is frequently found on the hands, arms, and legs, but it may develop on any part of the body.



FIG 688—CHILD WITH GENERAL GRANULOMATOUS ERUPTIONS AND MOTHER WITH THE PRIMARY LESION (FRAMBÆSOMA) ON THE LEFT MAMMA

The primary lesion or frambæcoma may heal before the general eruption begins, but, as a rule, is still present when the secondary eruption appears. We observed a case in which the primary lesion was still present six months after its first appearance, and when

In some cases the scar is small and smooth in others it is large and very thick. It is to be noted however that in Ceylon the disfiguring scar so frequently seen is partly due to the custom the natives have of cauterizing the sore deeply by very primitive methods. In other cases the large disfiguring cicatrix is due to the frambœsoma having developed on an old ulcer which on healing leaves a coarse scar.



FIG. 689.—FRAMBÆSIA GENERAL GRANULOMATOUS ERUPTION

**The Secondary or Granulomatous Stage**—The general eruption usually begins between one and three months after the first appearance of the primary lesion. It is preceded by malaise, headache, severe pains in the muscles, joints, and bones. In some cases there may be fever of an intermittent type. The patient, however, is ordinarily able to attend to his work. The general eruption develops as follows: minute roundish papules, the size of pin heads, appear on various parts of the body; some papules soon show a yellow point or minute yellow crust at their apex. Most of the papules



**FRAMBOESIA TROPICA**  
**GENERAL ERUPTION**



remain of practically the same size for many weeks and disappear leaving occasionally some furfuraceous patches others become larger several often coalescing and frequently acquiring a dark areola in natives a reddish one in Europeans Some of the larger papules increase in size and develop into the characteristic large

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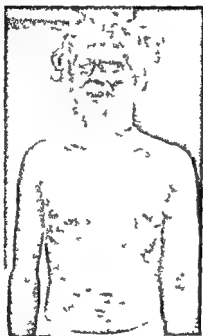


FIG 690 IRAMBOSIA (SECONDARY STAGE) GENERAL ERUPTION

slightly purulent secretion which soon dries into a crust These irambœtic granulomata are of various size from a large pea to a nut and may be found on practically any part of the body They are extremely common on the upper and lower limbs and on the face On the scalp they are very rare They may form rings round the mouth and anus and may enclose sound skin (so-called *yaus ringworm*) They may remain of the same size and appearance for months Often after a few weeks the secretion diminishes and a process of hyperkeratosis sets in They then become of much harder consistency and some of them especially those on the dorsum of the feet and toes may be covered with numerous small



hard, verrucose like protuberances. In the majority of cases—within three to six months in children and six to twelve months in adults—the granulomata dry up, shrink and disappear, leaving dark hyperpigmented spots or occasionally apigmented areas on their site which are most persistent. In some cases the granulomatous eruption may continue for several years, new crops of nodules appearing from time to time in succession. Each frambætic granuloma generally undergoes involution within two to four months leaving behind as a rule a dark area or, more rarely, a depigmented spot. Occasionally however, the granuloma does not regress so soon. In one of our patients a single granuloma persisted for two years after all the others had disappeared.



FIG 691.—FRAMBÆSIA GENERAL ERUPTION OF THE SECONDARY STAGE

The granulomata are seldom painful unless they develop between the toes on the soles of the feet or round the nails. They very often cause itching. The patient often exhales a peculiar offensive odour, which has been variously described as sour or musty. This is probably due to the growth of various bacteria representing

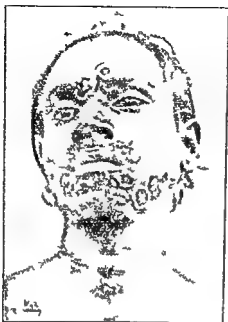


FIG 692—FRAMBÆSIA (SECONDARY STAGE) GENERAL ERUPTION



FIG 693—FRAMBÆSIA ERUPTION ON THE HANDS

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other types of erup  
s orally ulcerative



Γ 694 —FRAMBÆSIA CIRCINATA ERUPTION



Δ FIG 695 —FRAMBÆSIA GRANULOMATA ON THE HEELS OF THE FEET

At the same time several typical frambæsioid granulomata numerous small reddish papules with the epidermis intact other papules which have become moist and are covered by a tiny yellow crust

several furfuraceous patches here and there and spots of increased pigmentation at the place of previous granulomata. Occasionally



FIG 696—FRAMBESIA LESIONS ON SOLE OF FOOT



FIG 697—FRAMBESIA LESIONS ON SOLE OF FOOT

some granulomata break down and large irregular ulcers form. In the center reddish papillomatous masses which in our experience do not usually heal spontaneously. At times in

the latter period of the secondary stage peculiar roundish or irregularly outlined whitish patches are present especially on the back and arms with a nutmeg grater like surface. On closer observation these patches are seen to consist of numerous hard conical papules containing in their centre an epidermic plug which is easily removed leaving a depression in the papules. Sometimes the plugs are spiny and in this case the eruption closely resembles lichen spinulosus.

**ERUPTIONS ON THE PALMS AND SOLES**—The granulomatous eruption very frequently attacks the soles of the feet. At first dark brownish or intensely livid spots appear the thick epidermis is gradually pierced by frambæcial nodules similar to those found in other regions of the body. This affection of the soles is very painful the natives of Ceylon call it *dumas*. Similar lesions may

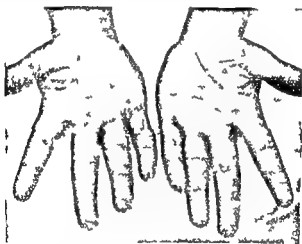


FIG 698—FRAMBÆSIA PITTED APPEARANCE OF THE HANDS

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cast off entirely though later they grow again

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After the granulomata have disappeared occasionally at the same time peeling whitish patches may be seen on the palms of the hands and soles of the feet closely resembling the syphilitic *psoriasis palmaris and plantaris*

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FIG 699.—FRAMBOESIA DACTYLITIS

base of the tongue also whitish patches closely resembling syphilitic leuoplakia. Small granulomata may develop on the nasal mucosa.

**CONSTITUTIONAL SYMPTOMS—Fever**—As already stated fever is frequently present, of intermittent or remittent type before the

is present The cervical and inguinal glands are most frequently enlarged

**ALIMENTARY SYSTEM**—As a rule the digestive functions are not disturbed

preceding the frequently four

preceding or concomitant malaria infection The microscopical examination of the feces of frambæsia patients will frequently reveal ova of various worms—*Ascaris lumbricoides* *Trichuris trichiura* and occasionally *Ancylostoma d iodenale*—but this is of frequent

well as the all granulo

matous ulcers are to be found in the nasal mucosa and more rarely in the larynx

**LOCOMOTORY SYSTEM**—*Joints*—In some patients several of the

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normal and

cause the swelling of the articulation to subside At other times one articulation only is involved and the symptoms may become so serious as to suggest purulent arthritis In many cases the smaller articulations become involved The symptoms in such cases are not acute and there is usually no fever

*Bones*—Inflammation of the periosteum of various bones is of

thel oma secondary carc noma myeloma and sarcoma The same author

*Muscles*—Contractures of various groups of muscles may be observed Fairly common is a contracture of the flexor muscles of the forearms This contracture is often permanent and in our opi  
ne

but also a true form of neuritis must be admitted We have seen in several cases clear symptoms of neuritis of the sciatic nerve with

severe pain along the course of the nerve, and signs of motor and trophic disturbances

**HYPERIDROSIS**—In several of our patients we have noticed hyperidrosis. The phenomenon was limited to the face in some cases to the hands and soles of the feet in others. It never extended to the whole body, and generally affected symmetrical regions. Hyperidrosis is more frequently observed in children than in adults. In a case at the Colombo Clinic a boy of fourteen presenting a

weeks or months

**THE EYES**—Granulomatous and papular eruptions may develop on the eyelids. A slight periostitis of the orbital margin is not rare, the margin becoming thickened and very painful on pressure. The occurrence of iritis is denied by most authors. In the Colombo Clinic two typical cases were observed during the general granulomatous eruption. In both cases the affection was of moderate severity. There was photophobia, ciliary congestion, discoloration of the cornea. P

secondary stage, papular and granulomatous, frequently involve the skin of the penis and of the labia. Granulomatous ulceration may be found on the vaginal mucosa. The urine, as a rule, does not contain anything abnormal, only when there is fever—as, for instance, when the articulations are acutely involved—then a slight amount of albumen may be present.

**THE BLOOD**—There is often a certain degree of anæmia, never very severe. The number of red blood corpuscles varied in our cases from 3,000,000 to 4,000,000, the hæmoglobin index (Fleischl) from 50 to 75. The red blood corpuscles did not show anything abnormal in their shape. On several occasions a comparatively

sees in syphilis, in which the lymphocytes are increased in number, while the large mononuclears are in normal amount. In many cases the eosinophiles are increased, this being probably due—in



part at least—to the presence of intestinal worms as revealed by the microscopic examination of the stools which shows frequently ova of *Ascaris lumbricoides* *Trichuris trichiura*, and in a few instances of *Ancylostoma duodenale*. Density and viscosity of blood seem to be normal according to Violle and the coagulability is not impaired. Auto agglutination is generally absent but has been recorded in certain cases.

**Complement Fixation Reactions**—Wassermann reaction is positive but is fairly often negative. Violle in frambæsia there if syphilitic liver is used as antigen while there is no fixation if an aqueous extract is used. In syphilis there is generally complete fixation.

**Cult. Reactions**—The cuts react on with frambæsia prepared with cultures of *T. pertenue* according to the technique used by Noguchi. In the preparation of luetin is often positive. Luetin also may at times give a positive reaction though less marked.

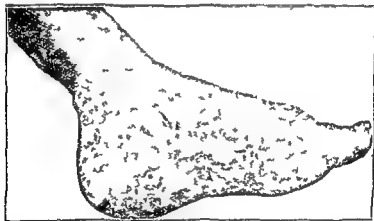


FIG. 60.—FRAMBÆSIA I. SUDO MACEGOMA

**CEREBRO SPINAL FLUID**—The liquid is in all cases perfectly clear like distilled water. No cellular sediment on centrifugalization is found in most cases. In a few some rare mononuclear cells are

fluid. No cholin is found. The reaction of the fluid is alkaline. The liquid is sterile; no treponemata can be detected.

**Tertiary or Late Stage**—The disease often terminates with the secondary stage. In some cases however the infection does not become extinct and tertiary lesions appear. These have been

denied by many observers but having been able to watch cases for several years through the whole course of the disease we have no doubt as to their existence. Sometimes the secondary and tertiary stages merge into each other but frequently the tertiary symptoms appear after the lesions of the secondary stage have undergone complete involution. The interval of time varies considerably in length and may extend to many years. The characteristic lesions of the tertiary period are gummatous like nodules and deep ulcerative processes. These gummatous nodules may develop in any tissues. When developed in the skin and subcutaneous tissues they are indolent and by their softening and breaking down ulcers are produced which may occasionally present clear-cut margins and a granulating fundus and when several contiguous nodules break down serpiginous ulcers are left. In other cases deep irregularly shaped ulcerations with very thick and undermined edges are seen in others—and these are the more numerous—large fungating ulcers are present. On healing these various ulcers leave whitish scars which are often thick and disfiguring. Frequently the scar tissue undergoes retraction and thereby causes permanent contractures and disfigurement. Lesions of the osseous type are very frequent painful nodes developing under the periosteum of several bones ribs sternum etc and we are inclined to believe that *Gangosa* (p 1876) an ulcerative condition of the palate nose and pharynx is in reality a tertiary manifestation of yaws. In other cases a diffuse chronic periostitis is present altering the normal shape of the bones. Contractures of various groups of muscles are frequently seen. Tertiary affections of the internal organs and of the central nervous system seem to be rare. Cases of aniridia considered to be of frambœrial origin have been



1 701 — FRAMBŒSIA  
TERTIARY STAGE

observed. The malady does not appear to be hereditary, in fact, it is worth noting that, in contrast to syphilis, parents generally contract the malady from their children.

**Fourth Stage: Paraframbæcial Affections.**—Cases of tabetic symptoms and symptoms pointing to paralysis progressiva believed to be due to an old frambæcial infection have been placed on record by Harper and others.

**Communicability.**—Frambæsia is usually conveyed by direct contact from person to person. It appears, however, that the germ is unable to enter through the normal skin, and that there must be some pre-existing abraded surface, small wound, or ulceration. Women are frequently infected by their children, the primary lesion appearing often on the mamme. In the native women of Ceylon the primary lesion frequently develops on the skin



FIG 702—FRAMBÆSIA TERTIARY STAGE

patients. Ants also are occasionally seen to go on to the frambæcial

ulcerations as well as on to ordinary ulcers. In Nuttall's classical work on the role of insects as carriers of parasitic diseases several

that they did not harbour any treponemata either on their mouth organs or on their legs. On examination after feeding the majority presented coarse spirochaetes and a few of them also *T. perenne*. In another experiment flies fed on yaws material were placed on scarified spots over the eyebrows of several monkeys and kept there for two hours by means of strips of gauze smeared with collodion at their margins. One of the monkeys became infected. Sambon considers that a fly of the genus *Hippelates* plays a very important role in the dissemination of the disease in the West Indies.

P. — 1 7

distinct inasmuch as (1) patients suffering from syphilis may contract yaws and patients suffering from yaws may contract syphilis (2) monkeys successfully inoculated with yaws do not acquire any immunity against syphilis (3) mercury has practically no action on frambœsia.

Syphilis has a world wide distribution frambœsia on the other hand is restricted to certain parts of the tropics. Frambœsia is extremely common in Ceylon extremely rare in India. Syphilis is

universally. Daniels has made the interesting observation that in British Guiana frambæsia of late has disappeared while syphilis is still rampant. As regards clinical features frambæsia differs from syphilis by the following characters: the primary lesion is as a rule extragenital; the principal type of eruption is a papule which proliferates into a characteristic frambæsi-form granulomatous eruption. The difference between frambæsia and syphilis is most marked in children. The granulomata present a more diffuse plasma cell infiltration and their bloodvessels have no tendency to the thickening of their walls.

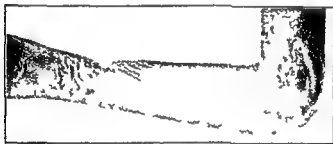


FIG 703.—FRAMBÆSIA TERTIARY STAGE

which is so characteristic of syphilis. Giant cells are generally absent. Naturally these differential histological details must be considered collectively as there is no individual histological character which exceptionally might not be present in both syphilis and frambæsia.

*Boubas and Pian*—Some of the older authors believed that under the names of yaws, boubas and pian three different diseases were indicated. All those however who have had opportunity to investigate frambæsia in different parts of the tropics have come

result inasmuch as he has been able to demonstrate that monkeys successfully inoculated with Ceylon frambæsia become immune to boubas and pian and vice versa.

Breda and De Amicis in Italy have not found *T. pertenue* in

Italian emigrants returning from Brazil and suffering from what they considered to be boubas but Splendore has shown their cases to have been cases of leishmaniasis and blastomycosis. The fact is that in South America the term boubas is used by the natives to cover several ethnically similar diseases while most medical writers use the term as a synonym for frambesia. Rivas Linderman and Robledo have found the *T. perenne* in their cases of boubas in Venezuela, Brazil and Colombia. It is not to be excluded however—in fact it is probable—that future investigation will show that there are several varieties of *T. perenne*.



FIGS 704 AND 705.—FRAMBESIA BEFORE AND AFTER TEN DAYS TREATMENT WITH CASTELLANI'S MIXTURE

*Cutaneous Leishmaniasis*—A type of cutaneous leishmaniasis (Bush Yaws) fairly common in the West Indies may simulate yaws but the presence of leishmania bodies and absence of the *Treponema perenne* will clear the diagnosis.

*Prognosis*—The prognosis is not serious so far as life is concerned. In 100% in the Central and South American cases.

ulcerated lesions becoming phagedenic, and giving opportunity to septicæmia and pyogenic processes to develop. Though framboesia rarely terminates in death, its long duration and great contagiousness render it a serious malady. The patients suffering from it are unable to attend to their work. Epidemics of fram-

LITTLE DATA SALVARSAN OR NEO SALVARSAN, while potassium iodide and tartar emetic are fairly efficacious and mercury practically-useless. Salvarsan and neo-salvarsan and their substitutes seem to act in framboesia more quickly and more powerfully than in any other spirochætal and treponemal condition, in fact in framboesia the *therapia sterilans magna* in Ehrlich's meaning, by a single dose can at times be obtained. Salvarsan was first tried with good results in experimental yaws by Nichols and in patients suffering from the disease by Strong in the Philippine Islands, and Castellani in Ceylon while Alston in the West Indies made the interesting observation that the serum of patients treated with salvarsan showed remarkable curative powers when injected in framboesia patients. Recently the salvarsan treatment of fram-

instead of salvarsan is generally used

MODE OF ADMINISTRATION AND DOSAGE —Neo salvarsan and its substitutes novarsenobenzol neokharsivan novoarsenobillon and to a certain extent galyl, are much more soluble than salvarsan and its substitutes arsenobenzol, kharsivan, etc. and are therefore used in practice in preference to salvarsan. Moreover, Castelli has shown that the *dosis tolerata* of neo-salvarsan in infected rabbits is nearly three times larger than for salvarsan, and that the *dosis sterilans* is one-tenth of the *dosis tolerata*.

The dosage of neo-salvarsan and most of its substitutes is, in

intravenous injection and we have found Ravaut's method of concentrated solutions very convenient, although we do not use

intravenous injection using a 10 c c syringe

The patient is made to lie down quietly on a couch or in bed. The skin is painted with tr. iod. and the veins of the bend of the

elbow made turgid by applying an elastic band round the arm

has been injected

Three to six injections of neo-salvarsan at three to six days interval are generally sufficient to obtain a cure though in a number of cases one injection is sufficient to make all the symptoms disappear. If one injection only is given 0.4 to 0.6 gramme should be injected when a course of three or more injections is carried out after 0.3 or 0.2 gramme the first time 0.2 or 0.1 gramme the second

heart the injection of neo salvarsan may be preceded by a hypodermic injection of caffeine. The patient may complain at times of headache and there may be a rise of temperature but very seldom are serious symptoms caused by the drug though cases of transient coma delirium epileptiform crisis nephritis and jaundice have been recorded

injection 0.4 gramme for men and 0.3 gramme for women being sufficient



lcerated lesions becoming phagedenic and giving opportunity to septicæmia and pyogenic processes to develop. Though frambœsia rarely terminates in death its long duration and great contagiousness render it a serious malady. The patients suffering from it are unable to attend to their work. Epidemics of frambœsia therefore are of the greatest consequence on tea sugar and other plantations as they reduce the supply of labour.

**Treatment**—The most efficacious and quickest treatment is by Ehrlich's Hata salvarsan or neo salvarsan while potassium iodide and tartar emetic are fairly efficacious and mercury practically useless. Salvarsan and neo-salvarsan and their substitutes seem to act in frambœsia more quickly and more powerfully than in any other spirochætal and treponemal condition in fact in frambœsia the *therapia sterilans magna* in Ehrlich's meaning by a single dose can at times be obtained. Salvarsan was first tried with good results in experimental yaws by Nichols and in patients suffering from the disease by Strong in the Philippine Islands and Castellani in Ceylon while Alston in the West Indies made the interesting observation that the serum of patients treated with salvarsan showed remarkable curative powers when injected in frambœsia patients. Recently the salvarsan treatment of frambœsia has become general having been used with very good results.

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tertiary

At the present time neo-salvarsan instead of salvarsan is generally used.

**MODE OF ADMINISTRATION AND DOSAGE**—Neo salvarsan and its substitutes novarsenobenzol neokharsivan novoarsenobillon and to a certain extent galyl are much more soluble than salvarsan and its substitutes arsenobenzol kharsivan etc. and are therefore used in practice in preference to salvarsan. Moreover Castellani has shown that the *dosis tolerata* of neo-salvarsan in infected rabbits is nearly three times larger than for salvarsan and that the *dosis sterilans* is one tenth of the *dosis tolerata*.

The dosage of neo-salvarsan and most of its substitutes is in

method of administration is by  
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0.4 grammes of neo salvarsan in 10 c.c. of sterile distilled water or sterile physiological salt solution and make the intravenous injection using a 10 c.c. syringe.

The patient is made to lie down quietly on a couch or in bed the skin is painted with tincture of iodine and the veins of the head of the

elbow made turgid by applying an elastic band round the arm

has been injected

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before the injection of neo-salvarsan may be preceded by a hypodermic injection of caffeine. The patient may complain at times of headache, and there may be a rise of temperature, but very seldom are serious symptoms caused by the drug, though cases of transient coma, delirium, epileptiform crisis, nephritis, and jaundice have been recorded.

sodium hyalurate solution for 0.6 gramme of salvarsan, a suspension of the drug in olive-oil or some other fatty material may also be used. A good preparation of this type which we have often used is by Pasini.

The suspensions in oil may often be given with advantage subcutaneously in the interscapular region. The intramuscular or subcutaneous injection of salvarsan, especially the acid solution, is generally painful and is followed by a hard infiltration which lasts for some weeks. Occasionally a slough forms which has to be removed surgically.

*Intravenous Injections*—The dose is smaller than for the intramuscular injection: 0.4 gramme for men and 0.3 gramme for women being sufficient.

ulcerated lesions becoming phagedenic and giving opportunity to septicæmia and pyogenic processes to develop. Though frambæsia rarely terminates in d tagiousness render it a ser from it are unable to attent boesia other

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Ehrlich Hata salvarsan or neo-salvarsan while potassium iodide and tartar emetic are fairly efficacious and mercury practically useless. Salvarsan and neo-salvarsan and their substitutes seem to act in frambæsia more quickly and more powerfully than in any other spirochætal and treponemal condition in fact in frambæsia the *therapia sterilans magna* in Ehrlich's meaning by a single dose can at times be obtained. Salvarsan was first tried with good results in experimental yaws by Nichols and in patients suffering from the disease by Strong in the Philippine Islands and Castellani in Ceylon while Alston in the West Indies made the interesting observation that the serum of patients treated with salvarsan showed remarkable curative powers when injected in frambæsia patients. Recently the salvarsan treatment of fram

good results

The sal

Relapses

however occasionally occur. In very old cases with tertiary lesions the treatment may fail. At the present time neo-salvarsan instead of salvarsan is generally used.

MODE OF ADMINISTRATION AND DOSAGE.—Neo salvarsan and its substitutes novarsenobenzol neokharsivan novoarsenobillon and to a certain extent galyl are much more soluble than salvarsan and its substitutes arsenobenzol kharsivan etc and are therefore used in practice in preference to salvarsan. Moreover Castelli has

shown that the *dosis tolerata* of neo-salvarsan in infected rabbits is three times larger than for salvarsan and that the *dosis* is one-tenth of the *dosis tolerata*

dosage of neo-salvarsan and most of its substitutes is, in

— 229 P

intravenous injection and we have found Ravaut's method of concentrated solutions very convenient although we do not use such highly concentrated solutions as does Ravaut who recommends dissolving neo-salvarsan in only 1 or 2 c.c. of water. We generally dissolve 0.3 or 0.4 gramme of neo-salvarsan in 10 c.c. of sterile solution and make the

on a couch or in bed

The skin is punctured with trepan and the veins of the bend of the

elbow made turgid by applying an elastic band round the arm

has been injected

Three to six injections of neo-salvarsan at three to six days'

be injected when a course of three or more injections is carried out  
 \* 0.05 gramme the first time, 0.1 gramme the second

heart the injection of neo salvarsan may be preceded by a hypodermic injection of caffeine. The patient may complain at times of headache, and there may be a rise of temperature, but very seldom are serious symptoms caused by the drug, though cases of transient coma, delirium, epileptiform crisis, nephritis and jaundice have been recorded

*Salvarsan* —Salvarsan may be given by intramuscular, subcutaneous, or

we have no personal experience

in olive-oil or some other fatty material may also be used. A good prepara

injection 0.4 gramme for men and 0.3 gramme for women being sufficient

The preparation of the solution to be injected is as follows. One of the glass phials in which salvarsan is put up in the dose of 6 grammes is broken and the contents (6 grammes of salvarsan) is carefully added to 30 or 40 c c

normal saline. 1 or 2 more drops of sodium hydrate may be required if the liquid is not clear. Each 50 c c of this solution contains 1 gramme of salvarsan. In man 200 c c should be injected in women 150 in children less according to the rules given *supra*.

For the intravenous injections special apparatus have been devised based on the principle of the Grantly douche. A convenient one which may serve for all these forms of injection has been placed on the market by W. H. Martindale.

**PRECAUTIONS TO BE OBSERVED**—The solution should be prepared with sterile salt solution made with freshly prepared distilled water and chemically pure sodium chloride. It should be perfectly clear. It should be slowly infused into

**Tartar Emetic**—Brodie in 1910 and later on other observers, tried antimonial preparations by intravenous injection as in sleeping sickness. The results are much less satisfactory than with salvarsan or neo-salvarsan.

**Intravenous Injections of Tartar Emetic associated with Other Drugs**—Potass iodide and mercury were associated by one of us with tartar emetic but the mercury did not seem to increase the action of tartar emetic. Here with two formulæ—

1	Tartar emetic	gr iiii
	Potass iodid	gr xxx
	Aq dest	ad ʒi
2	Tartar emetic	gr iiii
	Potass iodid	gr xxx
	Hydrarg perchlor	gr ʒi
	Aq dest	ad ʒi

One to 3 c c may be given diluted in 8 or 10 c c of sterile water by intravenous injection every other day.

**Treatment by Oral Administration of Drugs**—When neo salvarsan salvarsan or their substitutes are unobtainable, or in districts where lack of medical men and skilled nurses makes any method of treatment by injections difficult or impossible, treatment by oral administration is very convenient, and the mixture known in the tropics as 'Castellani's yaws mixture' will be found effective in many cases.

This mixture contains tartar emetic gr 1, potass iodid, ʒi, sodium salicylate, gr x, bicarbonate of soda gr xv, water or chloroform water, to 1 oz. One ounce is given three times daily of water, to adults and ʒi to children of eight less to younger children.

and decreases the emetic properties of the mixture in this way rendering possible the administration of massive doses of potassium iodide and large doses of tartar emetic. In the few cases in which emesis is produced the bicarbonate may be increased or a small amount of liq. morphine or codein given before each dose and in the comparatively rare cases in which severe iodism appears epinephrine as suggested by Milian in grm 0.002 doses may be given by the mouth or by subcutaneous injection twice daily.

The mixture as set down is cloudy although it becomes clear when diluted with water at the time of administering it. At the suggestion of Dr Dawson Williams some experiments were carried out to obtain a clear mixture and it was found that the addition of sodium tartar gr ʒ or of glycerine ʒii or of syrup ʒi per dose was sufficient to keep the mixture clear for weeks. The modified formula of the mixture is therefore as follows—Tartar emetic

satisfactory in recent and fairly recent cases when they may be compared with those obtained by the salvarsan treatment. In chronic cases the results are not so striking but as a rule much better than with any other known treatment except salvarsan or neo-salvarsan.



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 Trop Diseases



absent in patients suffering from . . . . .

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Barton's results as to the presence of a skin eruption in animals inoculated by these strains. They therefore conclude that Carrion's fever is a separate pathological entity distinct from verruga, and that it is a fever belonging to from the usual types by being occurring in patients suffering are supported by the clinical observations of several observers, among whom may be mentioned Eder

The next question which must be considered is the nature of verruga peruviana, for it has been suggested that it is frambæsia, and this has been specially emphasized, since it has been realized that it may run its course without any fever. Biffi, however, has clearly shown that they are separate, frambæsia being contagious, verruga not, frambæsia beginning with an initial lesion, verruga not, frambæsia being due to *Treponema pertenue* Castellani, verruga not. We may therefore conclude that verruga

verruga peruviana

can be inoculated into monkeys, it resembles closely Bassewitz's

**Climatology.**—Verruga peruviana is confined to South America, and to the western slopes of the Andes in Ecuador, Peru, Bolivia, and the northern parts of Chili, the most important endemic area being Peru, where it is almost limited to the departments of Ancachs and

and Cajatambo, which are on the western slopes of the Andes are most affected, while that of Santa, which runs along the littoral, is almost free. The disease is, however, peculiarly limited to certain places in these provinces. The heights of these places vary from Cochabamba, in the province of Cajatambo, which is only at an elevation of 406 metres, to Cajatambo, in the same province, which is at an altitude of 3,350 metres, but according to Monge it is never naturally

In the department of Lima there are six provinces, of which Chancay, Canta, Huarochiri, and Yauyos possess endemic areas, which vary in height from 900 to 2,030 metres. In Chancay there is Huaycho, in Canta, Acos, Viscas Yaso, Magdalena, and Yangas, in Huarochiri Santa Eulalia, Palle, San Gerónimo San Pedro de Casta, Surco Cocachacra, Santa Ana and Sisicayra, and in Yauyos, Omas

The height is more or less that which has along the river from

de Mama, Santa Ana, Corcóna Cocachacra, San Bartolome (where the stream which runs into the Cocachacra River is called the Aqua de Verrugas, because the people believed the disease came from drinking the water), Cuesta Blanca, and Surco

Here again as in the department of Ancachs the disease is restricted to certain deep, narrow valleys locally known as 'quebradas,' along which streams flow, and which are some 28 to 60 kilometres distant from the littoral, where the disease never occurs. A very important epidemiological point is that the disease only occurs in the summer when the rivers are in flood, and when all sorts of insects abound. Monge points out that earthquakes are

suggested that it was an intoxication due to sulphuretted hydrogen liberated from the earth, a view which never received any marked support. Odriozola and Tamayo have failed to find any bacteria in people suffering from the eruption of Peruvian wart. It is said to occur in animals especially quadrupeds, and not to be contagious. Chastang believes that some germ is inoculated by the thorns of *Cactus opuntia*. Long ago Raymondi suggested that verruga would, like rabies and syphilis be found some day to be due to a definite virus. Translating Raymondi's views into modern thought, it would mean that the three diseases would be found to be

due to a parasitic protozoon and indeed there is no doubt about the truth of this with regard to syphilis and with regard to rabies and it is quite possible that verruga peruviana will some day be found to be of protozoan origin thus confirming Raymond's striking conjecture

The study of the distribution of the disease in the various provinces of Peru as detailed above with the aid of a large-scale map will impress the reader with the similarity to the distribution of Rocky Mountain fever and will lead him to the conclusion that the

taken as mere suggestions as at present there is no direct evidence in favour of them Very minute bacillary like rods thicker in the

ence Similar bodies have been seen by De Vecchi Bassett Smith and Martin Mayer De Vecchi considers them to be products of degeneration

Strong Tyzzer Brues Sellards and Gastiaburu's experiments would point to the virus being a filterable one and inoculable in monkeys Inoculated in the testes of the dog and rabbit it induces characteristic changes As already stated these authors believe that the disease is not connected with Oroya fever which according to them is due to *Bartonella bacilliformis* (p 502) and is not inoculable in monkeys

Age sex and race appear to be of little importance as asserted that the colour of the eruption is due to mild attacks at a

the account of the pathology must be limited to very few remarks There has been great doubt as to whether verruga can be transmitted to animals by inoculation though Odrizola related that he inoculated a bitch with the blood from verruga lesions obtained from a post mortem with the result that the animal developed a typical skin eruption and eventually died The disease is said to occur naturally among animals—e.g. horses mules asses dogs and fowls—but especially among quadrupeds although Monge states that no one has definitely

ratory  
 ) some  
 illness  
 pains

in different parts of the body to be of the character of a septicæmia after which the typical eruption appears on the skin mucous membranes and internal organs when as a rule the general symptoms of fever etc abate from which one would infer that the organism had left the blood stream and become located in the skin and other organs. It is possible that it leaves the body by way of the skin. If however the local lesion develops in

of fever called Carrion's disease is produced.

The morbid anatomy is characterized by marked pallor of the

lips gums palate tongue pharynx larynx trachea cesophagus stomach small and large intestine in the substance of the liver spleen lungs thymus thyroid testicles kidneys and lymphatic glands and at times in the leptomeninges the choroid plexus the choroid coat of the eye in the substance of the muscles on the periosteum of bones on the peritoneal coverings of organs and on

is the reaction of the areolar tissue to some perivascular irritant. The connective tissue fibres become swollen and between them lie embryonic connective tissue cells while the

primary layer of the cutis has disappeared and the dermis proper is infiltrated with round cells which are mostly mononuclear or

cellular infiltration is very vascular and in the case of the older

tumours almost cavernous in structure, hence the liability to hæmorrhage, which is such a marked feature of the disease

The subcutaneous fatty tissue is always inflamed. In addition to str

may number 0.5 per cent, and there may be some large mononuclear cells like macrophages. The nuclei of the polymorphonuclears are simply bilobed

**Symptomatology.**—The incubation period is not definitely known, and is stated to vary from eight to forty days but to be most usually from twenty to thirty days, during which time prodromata, in the form of malaise lassitude, and depression, may be experienced

**Febrile Stage (Oroya Fever)**—The invasion is gradual, the prodromal symptoms increasing in virulence, while anæmia becomes apparent, and peculiar rheumatoid pains appear in different parts of the body. These pains are very striking and very misleading, for they may in some cases be limited to a single region, or even to a single joint or muscle, on the other hand, they may be more extensive, and lead to a diagnosis of some nerve disorder. As a rule, but not invariably, fever appears, and varies in intensity with the severity of the attack. There is usually insomnia and often delirium. Usually it is intermittent in character the paroxysm beginning about noon with chills, severe pains, much thirst, and a rise of temperature to about

rapidly becomes very anæmic and feeble, and usually constipated, but may at times suffer from severe diarrhœa. The destruction of red cells, according to Monge, is enormous, the number falling to 900,000 per cubic millimetre, with microcytes in large numbers,

leucocytosis, the count rising to 20,000 per cubic millimetre after the first few days, and increasing later. The polymorphonuclear leucocytes number about 75 per cent. The condition of the bone-marrow has been studied by Corvallo, who finds excess of normoblasts and neutrophile myelocytes

**Eruptive Stage (Verruga, sensu stricto)**—In many cases, after the febrile stage has lasted from twenty days to eight months, the skin begins to itch, and an eruption appears on the face, neck, the extensor surfaces of the arms and legs, and at times on the conjunctivæ, the lips, tongue, gums, palate, and pharynx. This

eruption shows itself at first as small, pinkish red, erythematous spots, sometimes associated with small vesicles, or more rarely with bullæ or pustules. The erythematous areas speedily become papules, and finally nodules which may vary in size and in number. The usual size is about that of a pea.

When fully developed they appear as elevated, cylindrical, fungiform or irregular wart like bodies, usually discrete, red in colour, generally firm to the touch (though they may be soft), and very liable to bleed. This type of eruption is the *forme miliare* (miliary type) of the Odrizofas and Salazar. In addition to these superficial tubercles there are deep subcutaneous nodules (nodular type), which lie under the unaltered skin, and from which at first they are quite free. These nodules may reach a large size, and become adherent to the skin, ulcerate and reach the surface as large red fungating masses which readily bleed. This is the *forme miliare* of the above mentioned authors. Both types appear on the skin, but the miliary type may also appear on the mucous membranes and internal organs while the nodular type is confined to the skin, especially at the flexures of the elbows and knees. The first crop usually appears on the face, and the extremities may be discrete or confluent, in the latter case no healthy area of skin may be visible. The miliary eruption may appear when the general symptoms have abated but the nodular is accompanied by fever.

The area of the skin on which the spots appear is usually œdematous, a feature most commonly observed on the legs. With the appearance of the eruption the fever declines the general

is raised. The white morphonucleosis. The protoplasm. At this

age the verrugas may develop in the internal organs, and cause serious symptoms, thus in the larynx they will cause dyspnoea, in the bronchi, bronchitis, in the lungs, pneumonia, in the pleura, pleurisy, in the nose, epistaxis and difficulty in nasal breathing, in the œsophagus, dysphagia, in the intestine bloody diarrhœa, in

appear and disappear, each preceded by an attack of fever, the eruption finally disappears, and the nodules, becoming pale and drying up, disappear without producing a scar, while the ulcerated nodules dry up and heal by cicatrization, and the patient is left

## CHAPTER LXIII

# RHINOSPORIDIOSIS AND SARCO- SPORIDIOSIS

Rhinosporidiosis—The Sarcosporidiosis—Sergentelliasis—References

### RHINOSPORIDIOSIS.

**Definition.**—Rhinosporidiosis is a chronic infection caused by *Rhinosporidium seeberti* Wernicke, 1900 and characterized by the production of polypus on mucous membranes and papillomata on cutaneous surfaces.

**History.**—The disease was first recognized by Malbran in South America in 1892, then by Seebert in 1906 in Buenos Aires, in a nasal polypus occurring in a young man aged nineteen years. In 1900 he gave a description of the parasite and its development, which we have been unable to obtain, but which is said to be a most excellent account. Later he found two other cases in the same town, and in 1900 the parasite was named *Coccidium seeberti* by Wernicke.

In 1903 Kinealy reported to the Laryngological Society a peculiar case of a polypus which he had found in 1894 growing from the

by Minchin and Fantham who came to the conclusion that the

by Beattie in 1906

In 1910 we observed the same parasite in a nasal polypus in Ceylon, and in the same year Ingram published an account of its occurrence in a conjunctival polypus and in a papilloma on the penis.

In 1914 Tirumurti gave a most excellent account of the disease.

In 1918 Chelliah, in Ceylon, not merely confirmed our original discovery of the disease in that island but reported several more cases in Singhalese and moormen.

**Climatology.**—Rhinosporidiosis occurs in South America, in India, and in Ceylon, and quite possibly in other regions.

**Etiology.**—The cause of the disease is *Rhinosporidium seoberi*

mature they are filled with pansporoblasts which have formed spore morulae containing some fourteen to sixteen clear shining spores.

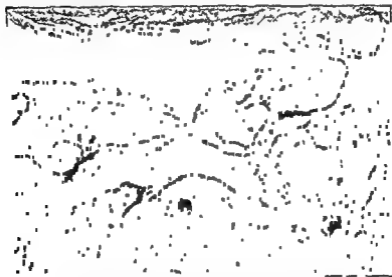


FIG 708—SECTION OF NASAL POLYPS SHOWING *Rhinosporidium seoberi* AT 1 AND 2 (X30) CEYLON CASE (PHOTOMICROGRAPH)  
1 IS REPRODUCED MUCH MORE HIGHLY MAGNIFIED IN FIG 709

The cyst ruptures, the pansporoblasts escape and rupture and so allow the spores to be liberated. Spores probably escape from the nose and other parts and possibly infect man in this way, because there is some slight evidence of transference direct from man to man, though we were unable to trace any such cause in our Ceylon case. Probably the reservoir for the parasite is in some unknown animal. Spores which do not escape from the body propagate



## CHAPTER LXIV

# PARAGONIMIASIS

Synonyms — Definition — History — Climatology — Aetiology — Pathology —  
Morbid anatomy — Symptomatology — Complication — Diagnosis — Treat-  
ment — Prophylaxis — References

**Synonyms** — Parasitic hæmoptysis, Pulmonary distomatosis, Endemic hæmoptysis

**Definition** — Paragonimiasis is a chronic or subacute general or local infection of man by means of *Paragonimus ringeri* Cobbold 1880 which produces cystic lesions containing a thick opaque reddish fluid in which are found at times the parasite or its eggs

**History** — In 1879 Ringer discovered the parasite of this disease in a patient at Tamsui in Formosa and it was named *Distomum*

and as Kerbert had named his species *Paragonimus westermans* in 1878 this name was applied to the human parasite until Ward and Hirsch stated that the spines which cover the cuticle and which are arranged in groups are different in the two species. Thus *Paragonimus ringeri* has chisel shaped moderately heavy spines while *P. westermans* has lancet shaped and very slender spines. The human species is therefore known by Cobbold's name of *P. ringeri*.

In 1880 Baelz found bodies in cases of hæmoptysis which he thought were psorosperms and therefore he called the disease gregarinosus pulmonum but when the bodies were shown to Leuckart he said that they were ova of a distomum

This Portuguese died in Formosa and Ringer discovered in the lungs during the post mortem examination a minute fleshy oval body grey in colour. This specimen was forwarded to Manson

of the disease as seen in the Philippine Islands. In 1910 Nagano reported upon the prevalence of the disease in Northern Formosa, around the prefecture of Shunchika, and Nakagawa in 1913 and 1914 found 1,249 cases, of which 922 occurred in that prefecture,

lands the cases were less in number . . .

*Uvulina* Gould which lives in pools and sluggish streams, in *Melania obliquegranosa* Smith, which inhabits slowly moving streams, and in *Melania tuberculata* Mueller.

The life history would be as follows.—The miracidia attach themselves by means of suckers to the head, jaws and feet of these molluscs, and then bore their way by means of their proboscis into the liver, the heart and the kidneys, where they become sporocysts and cercaræ. These latter possess an unforked tail and measure

Edwards and *Potamon (parahelphusa) sinensis* Milne Edwards

Dogs fed upon these crabs showed eggs in ninety days after

crab in the case of the infection of man, which apparently can take place via the skin

Nakagawa in

1914

Corca Japan,

Formosa, the Philippine Islands, and Sumatra. The infection is more prevalent among people living along the coast.

are done, in which the cercaræ are developed, and these pass to man either directly or through the agency of certain crabs in which they become encysted. Infection may be by the alimentary canal,

and also perhaps by the skin. The worms become adult in the lungs and other organs.

The disease appears to be very widespread in certain districts, and the old idea that it is more common in males than in females

Musgrave has classified the lesions into —

- 1 The non suppurating lesion
- 2 The tubercle-like lesion
- 3 The suppurating lesion
- 4 The ulcerative lesion—
  - (a) in the skin,
  - (b) in the bronchial mucosa
  - (c) in the intestinal mucosa,
  - (d) in the bile-duct

tis.  
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formation of a cirrhosis or a round celled infiltration, with some times many eosinophiles, which may lead to abscess formation, and finally to ulceration. The abscess formation may at times produce caseous material, giving a tubercular appearance.

The non suppurating lesion may therefore be simple infiltration

with the presence of eggs in brown patches at the points of contact. The inflammation of the organ may, however, proceed to pus formation, resulting in a typical abscess. On the other hand, the tissues generally attempt to circumscribe this abscess by a fibrous wall, and thus produce what is called the typical lesion of the disease. In the centre of the abscess will be seen degenerated cells, blood, eggs, and perhaps a parasite. Then comes a capsular wall

parasite, are formed. These lesions may work their way to a cutaneous or mucous surface and so open into a bronchus, or into the intestine, or the bile duct, or on to the skin, thus giving rise to ulcers in those regions. The intestinal ulcers closely resemble dysenteric ulcers, and indeed, may become secondarily infected with amœbe or bacteria.

In course of time these lesions may show some attempt at healing and scar-formation, but generally this is not very successful.

The blood may show a deviation of complement with worm body used as antigen.

**Morbid Anatomy.**—The body may be well nourished, but more

hepatitis, and abscess formation. The typical lesions mentioned above may be found in the spleen, pancreas, small and large intestines, kidneys, bladder, epididymis, prostate, and in the choroid plexus of the brain

crystals. The patient generally complains of pain in some part of the chest. The physical signs may indicate broncho pneumonia or pleural effusion which may be serous or purulent.

In the abdominal form of the disease there are only the vaguest symptoms of dull aching pains and tenderness while diarrhea,

present

The blood and urine have not yet been fully examined. Though generally chronic the course may be acute if complicated by septic or other diseases.

**Complications.**—The most usual complications are tuberculosis

or the fluids obtained by puncture of a hydro or pyothorax. Suspicion as to the presence of the disease will be strongest when cases showing any of the above signs occur in the endemic area.

**Treatment.**—The treatment must be devoted to relieving indi-



## CHAPTER LXX

### KATAYAMA DISEASE

Synonyms — Definition — History — Climatology — Etiology — Pathology —  
Morbil anatomy — Symptomatology — Varieties — Complications — Diag-  
nosis — Prognosis — Treatment — Prophylaxis — References

**Synonyms** — Urticarial fever Asiatic schistosomiasis Schistosomiasis japonica  
Kabure (cutaneous symptoms)

**Definition** — Katayama disease is caused by *Schistosoma japonicum*  
Katsurada 1904 and is characterized by urticarial and dysenteric  
symptoms painful enlargement of the liver and spleen with or  
without fever drowsy progressive anemia and sometimes pulmo-  
nary and brain symptoms

**History** — In 1887 Mazumi drew attention to a peculiar form of  
cirrhosis of the liver which was found in certain districts in Japan  
and was caused by the ova of some unknown parasite. His dis-  
covery was confirmed and the ova were found in other organs

adults in the portal vein of a cat. He named the parasite  
*Schistosoma japonicum*. In the same year Fujinami discovered a

1911 Houghton Logan and Lambert drew attention to cases of  
fever with urticaria and eosinophilia connected with infections with  
*S. japonicum*

In the same year Edgar drew attention to this fever in the Yangtze  
Valley near Hankow and noted that nearly every patient had  
bathed or waded in marshy ground near the river

In 1912 Miyagawa did not believe that the worm was the cause of  
the dermatitis

In 1913 Miyagi and Suzuki noticed that the eggs of the worm,  
when kept for one to two hours in feces and water at a suitable

with these snails for three hours a day develop *S japonicum* in their livers

Also in 1913 Katsurada found that the worms reached maturity

..

suffered from fever about 102° to 103° F in the evening and normal in the morning pains in the lumbar and epigastric regions pulse-rate 90 with a temperature of 103° F slight reduction of the red corpuscles 4 800 000 per c mm and 50 per cent to 70 per cent of eosinophiles and loss of weight associated with the appearance of urticarial eruptions in various parts of the body

In the same year Lanning noted that it was not uncommon for a fair proportion of the crews of gunboats patrolling the Yangtze River to become infected after wading through the water covered paddy fields in search of snipe

Miyuri's work induced Leiper and Atkinson in 1914 to proceed to Shanghai and later to Katayama in Japan to investigate the parasite Their results were published in 1915 At Katayama they found a small brown snail with eight spirals and an operculum known at that time as *Katayama nosophora* which had an extraordinary attraction for the miracidia its small head and foot becoming festooned with white specks (the miracidia) which appeared to irritate the snail Later the liver was found to be full of cercariæ with bifid tails which infected laboratory bred mice by passing through the skin male and female adult worms being found in the portal vessels one month after infection

In 1916 Koiki drew attention to the fact that in forty two cases found near Shushin in Japan all but three were farmers and most

only the low lying lands appear to be infected no cases from the hills or mountains are known

vertebrate reservoirs are cats, dogs and pigs. For description of the worm see p. 590.

**Pathology**—After penetrating into the skin the parasites enter either veins or arteries. In the former case they pass to the right heart and hence to the lungs at the bases of which they collect and then passing through the mediastinum, diaphragm and liver reach the portal system. Suezasu in 1916 obtained complement fixation with the blood of immune animals.

**Morbid Anatomy**—On opening the abdomen signs of old peritonitis may be seen the appendices epiploicæ being matted together and at times there are also signs of old pelvic peritonitis. The liver is cirrhotic and less than its normal size and its surface is studded by nodules usually larger than those of alcoholic atrophic cirrhosis. Glisson's capsule is thickened and shows much connective tissue with round celled infiltration in which lie the ova of the worm.

The small and large intestines and appendix may be thickened and their mucosa is swollen and hyperæmic and



FIG. 111.—*Schistosoma japonicum* Katsurada (After Manson)

mesenteric glands, the wall of the gall

#### vessels

In addition the eggs may be found in fibrous and round-celled infiltrations in the lungs and in the brain. This infiltration often takes the form of nodules.

**Symptomatology**—The early symptoms of the disease may be slight or perhaps it may begin with attacks of fever with urticarial rashes in which there is marked eosinophilia and this may be associated with cough, scanty expectoration, some impairment of resonance over the bases or other parts of the lungs with fine crepitant râles on deep inspiration and a diminution of the breath sounds.

About two years later there are diarrhœic or dysenteric symp



toms, with or without fever, and the presence of the ova in the faeces. Associated with these symptoms are abdominal pains, the hypogastrium shrinking, giving rise to a characteristic time the dysenteric symptoms

may cease, and the organ may begin to shrink, but in any case the spleen becomes tender and enlarges, ascites appears, and the patient becomes steadily weaker, more and more anæmic, emaciated and incapable of mental or physical work. The average of three differential blood counts by Peake is as follows: Polymorphonuclears, 56.6 per cent, mononuclears, 13.2 per cent, lymphocytes 15.6 per cent, eosinophiles, 14.1 per cent, but the eosinophilia may reach 50 per cent. Attacks of fever may occur nightly otherwise the temperature may be subnormal. The vascular, respiratory, nervous, and urinary systems are usually normal. If however the ova affect the lungs, there may be signs of bronchitis, broncho-pneumonia and fibrosis, and if the brain those of Jacksonian epilepsy. In children the development is stunted. Death may result directly from the action of the parasite or be due to some intercurrent disease.

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either diarrhoea or constipation. The characteristic ova can be found in the motions

*Third Stage*—This may or may not be present and then only after three

**Varieties.**—Houghton recognizes the following types of the disease: (a) Typical cases, with enlarged liver and spleen, ascites, and blood in the motions; (b) cases with only splenic enlargement, and with or without blood in the motions; (c) cases with cerebral symptoms and marked eosinophilia to which may be added—(d) urticarial fever, with marked eosinophilia in the early stages.

The splenic type when present without blood in the motions, may give rise to difficulties of diagnosis, which may be cleared up by an examination of the blood and faeces. The eosinophilia in these cases is from 25 to 51 per cent.

The cerebral type is exemplified by partial hemiplegia and slight disturbance of speech after high fever and associated with an eosinophilia of about 50 per cent. Jacksonian epilepsy has also been reported as due to this parasite.

blotches on the arms trunk and legs and associated with a marked urticarial rash. The fever lasts some weeks and very closely resembles malaria at first because the daily fall of temperature is associated with sweating.

**Complications**—The infection is generally complicated by the presence of *Trichuris trichiura*, *Ancylostoma duodenale* or *Necator americanus* and *Ascaris lumbricoides*. Dysentery is a complication which may occur and prove fatal to the patient.

**Diagnosis**—The characteristic signs are chronic painful enlargement of the liver and spleen associated with ascites and chronic irregular diarrhoea and marked eosinophilia (10 to 50 per cent). A definite diagnosis is to be effected by finding the ova in the feces. These ova are large (0.1 by 0.07 millimetre) oval non operculated laterally spined (75 per cent) smooth and transparent with a double contour sometimes showing a *Miracidium* and when kept in water for a short time give rise to a free-swimming ciliated *Miracidium*. These ova are apt to be mistaken for *Ascaris lumbricoides* or less likely for an *Ancylostoma* ovum. The ova require to be looked for carefully.



FIG. 712.—EGG OF *Schistosoma japonicum*

(From a photomicrograph by J. J. Bell.)

Other points which assist in the diagnosis are the greatly exaggerated knee-jerks, the peculiar muddy complexion suggestive of anemia, the lack of leucocytosis (the counts in uncomplicated cases being about 2,000 to 8,500 per cubic millimetre) and the emaciation with out obvious cause.

**Prognosis**—The prognosis is very bad as the parasite directly or indirectly leads to the death of the patient. The mortality is not known but Katsurada met with between thirty to fifty-four cases every year for five years in the infected area in Japan and saw three to five deaths per annum which he considered directly due to the parasite—i.e. a mortality of about 10 per cent—but

he thinks that the indirect mortality would raise the percentage considerably

**Treatment**—The only treatment that can be suggested is to administer salvarsan or tartar emetic

**Prophylaxis**—Avoid contaminated water in drinking and bathing—*i. e.*, use boiled water in infected areas for both purposes. Wading in swamps lakes and paddy fields is very dangerous and is the method of infection of man

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## CHAPTER LXVI

# THE FILARIASES

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**Synonyms** Filarial disease French Filariose Maladies filariennes  
Ital an Filariasi German Filaria Kraokh it

**Definition**—Filariasis is a term denoting the infection of man or animals by any species of *Filaria*—e g *Filaria bancrofti* Cobbold 1877 and some others

### FILARIASIS CAUSED BY *FILARIA BANCROFTI* Cobbold 1877

The diseases produced in man by *Filaria bancrofti* Cobbold 1877 include lymphangitis orcl tis vari in lymphatics and lymphatic glands chylous and lymphatic extravasations and elephantiasis

**History**—The appearance of the huge leg of elephant is a so striking  
cl

The word elephantiasis as first used by Celsus to indicate leprosy, and in this sense is followed by most writers until Calen who included true

of the Arabs

In 1812 Chapotain was the first to describe hæmatochyluria in Mauritius and he was followed by Salese in 1832 whose paper aroused so much interest in Brazil where the disease had for long been well known that in 1835 a

diseases with elephantiasis

in the fluid from a case of chylous ascites

lymphatics of a lymph scrotum and from varicose lymphatic glands and suspected that the so called malarial orchitis must be of filarial origin. He also obtained a female worm from a case of elephantiasis of the scrotum and in 1879 Lewis found pieces of male and female worms while in 1888 Sibthorpe obtained perfect specimens of a male and female from a lymph scrotum the former being described by Bourne. Lastly in 1898 Maitland

ich he  
to be

phangitis of lymphatic abscesses of varices of lymphatics and of lymph

theory has not been supported by Fulleborn's observations on Samoan

and elephantiasis

The morbid anatomy has been investigated by but few observers notably by Mackenzie Manson Low Young and Bahr

**Climatology.**—The fact that ancient Indian writers were acquainted with elephantiasis of the leg and scrotum while Celsus does not appear to have known the disease and the further fact, mentioned by both Hillary and Henty, that elephantiasis was rare in Barbados at the beginning of the eighteenth century, together with Hillary's views that the disease was introduced into that island by negro slaves from Africa awaken suspicions that the

about 23° S in the western hemisphere

China,

and in  
Islands,

but is absent in the Sandwich Islands It is also known in New Guinea

In *America* it occurs in the Southern United States in Central America the West Indies in Guiana, Venezuela Brazil, Peru, and Columbia

In *Africa* it is common on the West Coast in South Africa, East Africa Madagascar, Reunion Mauritius Morocco and Egypt and Northern Africa

In *Europe* it is said to exist near Barcelona and in Turkey

In these countries its distribution is unequal being in general

both are common

Still more interesting is his observation that at the southern end of Lake Nyassa there was only one case of filariasis met with, and none of elephantiasis while at the northern extremity both were frequently seen

Low has also studied the distribution in the West Indies Here

again, this is very unequal, some of the islands—Barbados, for example—being heavily infected while in others, Grenada, the infection does not appear to exist.

The distribution must depend upon the presence or absence of mosquitoes capable of disseminating the worm, but this aspect of the epidemiology still requires a considerable amount of research, further, the distribution of the suitable mosquitoes must depend upon many factors concerning which we are quite ignorant. When these conditions are better understood the climatology will be

temperature and con-  
 ditions have been known to be  
 related to elephantiasis, and

the reason of this has recently been explained by Fulleborn's experiments on *Dirofilaria immitis*. This observer found as the result of most careful experiments that the *Dirofilaria* developed better in mosquitoes if the air temperature was high, and in that respect

to it

With reference to Fiji Bahr concludes that it is possible that at one time or another nearly every Fijian is the subject of filariasis because 27.1 per cent were found to harbour *Microfilaria* in their blood, and adult worms could be found in the lymphatics and other tissues, and others (25.4 per cent) were found to suffer from filarial disease when no *Microfilaria* could be found in the blood, and, lastly, in patients while still under observation the *Microfilaria* have disappeared from the blood.

*immitis* Theobald 1903, *Celia stomana* Wiedemann 1821, *Simulium*  
 " Theobald 1903, while the worm is known to  
 not in certain other

development in any and

After development in the thoracic muscles of the mosquito the

embryo passes into the labium and when the mosquito bites it

pletely filled by these appendages

Its further history and wanderings in the body are quite unknown until the adult condition is reached. The adults (males and females) are generally found lying together though the females appear to be in preponderant numbers in lymphatic vessels but they can also be found in the lymphatic glands while dead and calcified worms have been found not merely in lymphatic glands but also in the testes epididymis spermatic cord and tunica vaginalis. Here the female produces the thin *Microfilaria* which

### cycle of development

It is interesting to note that in various parts of the tropics natives believe that elephantiasis and other filarial diseases may be transmitted through sexual intercourse.

The adults lying in the lymphatic vessels may mechanically cause obstruction to the flow of lymph and thus produce varices inflammation of vessels and glands and if the varicose vessels rupture extravasation of lymph or chyle.

While this etiological relationship of the worm to the lymphangitis and lymphatic abscesses to the varices in lymphatics and lymphatic glands to hæmato-chyluria and chylous extravasations is admitted by all observers there are those who doubt this relationship with regard to elephantiasis. These authors base their objections upon the facts that the worm and its larvæ may be absent in well developed cases and that the disease can occur in countries in which filariasis is believed not to be present both of which are quite true but are capable of explanation. There is an undoubted general relationship between the number of cases of filariasis and of elephantiasis in a district. Where there is no filariasis elephantiasis is either extremely rare or unknown where there is abundant filariasis there are also many cases of elephantiasis.

In investigating this point in a locality care must be taken to exclude immigrant cases of both filariasis and elephantiasis. Thus, Low failed to find either condition in the inhabitants of the forests of British Guiana and in the Wandas natives of Uganda though immigrant cases were met with.



The adult *Filaria* has been found in the tissues removed by operation from a case of elephantiasis of the scrotum and further the condition of elephantiasis is produced as a rule by a series of attacks of lymphangitis which in every particular resemble undoubted filarial lymphangitis

It is true that a secondary bacterial infection may possibly assist the development of the disease for a diplococcus has been found by Dufogere which he calls the lymphococcus and his findings have been confirmed by Goulerton Le Dantec describes a similar organism which he calls the dermatococcus but the main cause of elephantiasis in the tropics is *Filaria bancrofti* though it is quite possible that exceptionally other causes may lead to occlusion of lymphatics and the formation of elephantiasis

**Pathology**—If the parent worms live in positions in which they do not obstruct the flow of the lymph and if they are not accidentally injured no pathological effects will be produced on the host and our observations support Manson's theory that the presence of the worms may produce no ill effect upon the host for we know of a case where for years they have produced no symptoms

But if the parent worms obstruct the circulation of the lymph mechanically—for example when three or four come together in an important main lymphatic trunk—then the retained lymph is certain by mechanical pressure to damage the tributary channels. Further if any accidental injury is inflicted upon the female parent worm this may cause abortion and as a result the production of oval eggs instead of elongated embryos (Fig 268) and these as will be explained below are liable to block up the small lymph channels of the skin or of a lymphatic gland. Therefore Low is quite correct in his statement that the *Filaria* is not entirely compatible with health for very slight causes will produce disease.

In certain districts from 5 to 27 per cent of the population is infected with filariasis and therefore if there are many mosquitoes

case is one of varicose lymph the obstruction the lacteals with the

milky it is obvious that the obstruction must be beyond this point

The cause of the obstruction may be a coiled up mass of worms—

with clear junction of the fluid is

*e.g.* Young found six females and one male in such a bundle—and they may be discovered behind a valve or in a dilated sinus. A single female worm may however be found lying in a dilated lymphatic the draining gland being probably blocked by the aborted ova. The irritation caused by the worms may lead to a permanent blocking of a main lymph channel which will persist even after the irritating worms have died and disappeared as has been observed by Mackenzie or again the thoracic duct may be found dilated in part of its course but quite patent throughout.

to engorgement of the renal the lumbar and the pelvic lymphatic channels with lymph as well as that of engorgement and dilatation of the lacteal vessels themselves.

If the lymphatic vessels of the bladder or other parts of the

the milky opacity is due to a large amount of protein and not to fat and this observation has been confirmed by Low who in one case found the lacteals normal and showed that the milky fluid was lymph proceeding from dilated lymphatics in the kidneys ureter and bladder. If the abdominal lymphatics rupture there will be chylous ascites if those of the tunica vaginalis there will be chylocele.

If not with the

by a damage to the

occur and as a result the connective tissue would become inflamed and hypertrophied which together with the excess of lymph would increase the size of the part. Manson bases this theory on his observation of eggs escaping from the ruptured vesicles of lymph scrotum. Bahr is of the opinion that tropical elephantiasis can best be explained by the blockage of the lymphatic channels of the diseased area by the frequent and long continued invasion of the adult *Filaria*. He finds that the *Microfilaria* may not reach the blood but die in the gland or organ in which they are lying. He also finds that the periodical discharge of these *Microfilaria* may be a factor in the production of lymphangitis, orchitis and funiculitis and that the parent worm may die after these inflammatory attacks.

It is believed that the smooth elephantiasis (*elephantiasis glabra*) in which the skin is smooth is due to blocking of the channels in the groin glands and rough elephantiasis (*elephantiasis verrucosa*) in which the skin is very nodular is due to blocking of the small skin capillaries but we are not acquainted with definite proofs of this theory.

*The Blood* —The blood in filariasis does not exhibit anemia unless there is hæmatochyluria or diarrhoea the number of leucocytes is normal but there may be leucocytosis during the attacks of fever. The eosinophiles are at times increased.

*Morbid Anatomy* —The morbid anatomy naturally varies with the variety of the pathological lesion produced.

In lymphangitis the lymphatic vessels will be found enlarged and inflamed and abscesses of varying size may at times be found containing the dead worms which are apt to become calcified by the deposition of lamellar plates of calcium carbonate in the interior of the worm. The calcified worms were first described by Wise as small yellow bodies with the shape and structure of *Filaria* which he found in the pelvis of the kidney. Bahr states that at a later

the groin and in the quadriceps extensor in the leg and over the internal condyle in the axilla in the latissimus dorsi and serratus magnus muscles in the arm. In these abscesses the dead worm was found associated with *Staphylococcus pyogenes aureus* and *Streptococcus pyogenes*.

In this manner the lymphatic becomes thickened but shows also numerous cyst like dilatations in which the dead worms may be found. The fugitive swellings found in filariasis have been proved by

Young to be composed of dilated lymphatic tissue. Inflammatory masses adherent to the skin in various parts of the body have been found to contain the adult worm.

In lymphatic varix or varicose lymphatic glands the obvious lesions may, and generally do, form part of a much larger dilatation of the pelvic and lumbar lymph vessels and glands. The vessels are found enormously dilated with thickened walls, while the

numerous compartments

In chylous extravasations the thoracic duct may or may not be found impervious, but in any case the lacteals, the lumbar, pelvic, pudendal, and crural lymph vessels will be found enormously dilated, and the lumbar lymph glands converted into septated sacs. The site of the ruptured lymphatics is, however by no means easy to find.

In elephantiasis the lymphatic vessels will be found dilated and thickened, and in early cases a round celled infiltration may be seen in the connective tissue of the part, but in later cases this has

may or may not be degenerate

In cutting into the tissue of a region affected with elephantiasis the skin may be noted to be thickened, and below it there will be found dense fibrous trabeculae, with the spaces filled with yellow, oily, fatty substance, which exudes lymph, while the vessels and nerves will be found much in retracted state.

### THE CLINICAL DESCRIPTION.

General Remarks—The clinical description of the various

## FILARIAL LYMPHANGITIS

Synonyms — Elephantoid fever Lihwa (Tijian term for a rig

Remarks — Attacks of lymphangitis associated with an erysipelatous eruption of the skin are extremely common in the tropics and are often of a filarial nature and by their repeated recurrence produce elephantiasis

Symptomatology — The attack often begins with a shivering and a rise of temperature to any degree from  $101^{\circ}$  to  $104^{\circ}$  F with vomiting and headache In some cases there is no pain in the

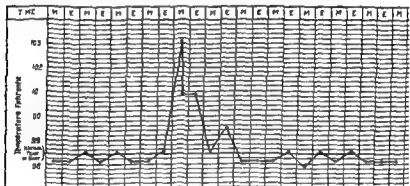


FIG 713 — TEMPERATURE CHART OF A CASE OF ELEPHANTIASIS OF THE LEG DURING AN ATTACK OF FILARIAL FEVER

affected area but a careful examination of the sensation of pain on the part of the patient will lead to the discovery of a red oedematous area of skin from which the inflamed lymphatic may be noted extending towards the nearest lymphatic glands which may or may not be inflamed and painful Usually the temperature falls quickly in a couple of days to normal and does not rise again though it may be several days before the erysipelatous rash disappears and the lymphatics return to normal

tal # 1109 THIS SUBJECT IS COVERED BY A B W O C Y  
it in all detail The history of the case will indicate the correct diagnosis

Treatment —  
tive and a litt

chloridi  $\mathcal{M} \times \mathcal{V}$  given three times a day well diluted. Locally

### FILARIAL ORCHITIS AND HYDROCFLE

**Symptomatology**—This complaint begins with pains in the testicle fever and at times rigor pains in the back and lower part of the abdomen and groins and bilio vomiting. The testicle enlarges and is tender and painful while an effusion forms in the tunica vaginalis of either lymph or chyle. The lymph thrown out is at first inflammatory and may coagulate and is usually absorbed after the fever subsides but may persist and form a filarial hydrocele. The effusion of chyle is however more usually permanent and forms one of the varieties of chylocele to be mentioned later.

**Treatment**—The treatment consists of rest in bed and the application of lead and opium lotion and cool applications together with fairly vigorous purgation of the bowels.

### FILARIAL LYMPHANGIECTASIS

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FIG 714—FILARIAL LYMPHANGIECTASIS

**Symptomatology**—In the scrotum the affection begins

discharge either lymph or chyle containing filarial embryos or much more rarely eggs

If the vesicles are ruptured the discharge of lymph may be quite considerable in the twenty four hours and may produce such

times with fever. When the acute symptoms have subsided a swelling like a varicocele which disappears in the prone position



FIG 715.—FILARIAL LYMPHANGIECTASIS

In lymphangitis of the leg there is the same fever with swelling of the glands leaving a soft swelling in the groin which disappears on lying down and reappears on standing up and which has no impulse on coughing

**Treatment**—As the local condition is simply part of a much more generalized varicosity of the lymphatic vessels it is best to treat it symptomatically with antiseptic dusting powders such as boracic acid zinc oxide and dermatol etc

may be followed by elephantiasis or even chyluria

### FILARIAL ABSCESSSES

Manson Low Wise Bahr and others have called attention to the frequency of filarial abscesses in various parts of the body. The most important are those found in the thorax and in the retroperitoneal tissues. In the latter situation the symptoms may be those of peritonitis



FIG 716—FILARIAL PHLEBECTASIS SIMULATING VARICOSE LABYRINTHIC GLANDS AND VARICOCELE PATIENT IN THE UPRIGHT POSITION



FIG 717—FILARIAL PHLEBECTASIS SAME PATIENT LYING DOWN (Photograph taken from above Note the disappearance of the swellings)

### FILARIAL PHLEBECTASIS (FILARIAL VARIX)

At times persons suffering from filariasis exhibit as observed by us marked varicose conditions of various veins. Occasionally in association with enlarged superficial vein large masses are to be



seen in the axilla groins and other regions which on superficial examination might be taken for enlarged glands but on palpation it can be ascertained that they are composed of veins the same sensation being obtained as that experienced when palpating a varicocele When these masses occur in the groins they disappear when the patient lies down

### VARICOSE LYMPHATIC GLANDS

**Synonyms** —*Helminthoma elastica* Adenolymphocele

**Definition** —Varicose lymphatic glands are glands enlarged some times to an enormous extent by dilatation of their lymph paths

the parotid lymphatic gland has been recorded to have been affected In post mortems as already noted the lumbar glands may be found converted into separated sacs

attacks of fever and  
which are easily movable  
in They are found  
in the groin inguinal or femoral regions and in the axilla If  
punctured with a hypodermic needle lymph or chyle can be  
obtained at times containing *Microfilaria* Usually small they  
may assume enormous proportions reaching below the knee and  
seriously impeding locomotion

**Treatment** —They may be removed if necessary but this should not be done without due cause as they are only part of a more widespread disorder Radium treatment has been advised by Sir Havelock Charles

### FILARIAL LYMPH AND CHYLOUS EXTRAVASATIONS

Lymph and chylous extravasations are due to the rupture of

Chyluria and Lymphuria Chylous and Lymphatic Diarrhoea  
Chylocele and Chylous Ascites Perhaps further investigations  
will show that Wise and Low are correct and that in addition to  
hæmato chyluria and other chylous conditions there may also be a  
pure hæmato lymphuria lymphatic diarrhoea lymphocele and  
lymphatic ascites

#### Chyluria and Lymphuria

**Pathology**—This has been worked out principally by Mackenzie and Manson and more recently by Low and Wise. The presence of chyle in the urine is due to a blockage of a lymphatic vessel with varicosity of lymphatics in the bladder walls so that the chyle passes into the bladder.

The condition takes place when the blockage of a lymphatic vessel is permanent. Chemically the chyle is

65111

**Symptomatology**—The onset of the attack is usually abrupt, without marked symptoms though vague pains may be felt and at times there may be fever pains in back perineum and thighs. Usually however the patient simply asks advice because he is

but it is merely an  
 recurrent and inter-  
 mittent. The  
 attack lasts for  
 days or years while  
 the patient is in  
 bed. After con-  
 valescence the urine  
 is dry.

The urine is  
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 and a  
 little  
 It  
 is the

the urine is  
 much  
 accumulated.  
 Under  
 the microscope  
 white are  
 and red  
 oxalate,  
 to 1020  
 ether, the

fat can be removed and estimated which is usually found to vary from 0.8 to 1.8 per cent. After the removal of the fat the urine can be tested for albumen which is always present and which usually persists for some time after the fat has disappeared. The quantity of albumen varies from 0.6 to 0.9 per cent. Albumoses and sugars are absent.

When the urine does not contain any distinct amount of fat one speaks of lymphuria and if blood is present hemato lymphuria. The



**Varieties** —The most common varieties of elephantiasis are the affection of the leg scrotum vulva arm and breast while other regions are much more rarely affected. The different varieties must now be briefly described.

### Elephantiasis of the Leg

**Symptomatology** —During attacks of filarial lymphangitis of the leg it is noticed that the limb becomes swollen and though after the first attack it may resume its normal size this does not happen after repeated successive attacks and gradually the limb becomes



FIG 718 —ELEPHANTIASIS OF THE LEGS

swollen and puffy, and is separated by the deep ankle sulcus from

the swollen lower part of the leg. In these sulci the debris of the cast-off epithelium, together with the excretions of the skin, accumulate and give rise to a foul-smelling discharge, and ulcers may form. At first the skin is smooth and soft, forming the smooth variety of the complaint, which may persist, or it may become dark, hard, thick, and rough, being elevated into bosses or warty elevations, forming the verrucose variety. The appendages now atrophy



FIG. 719.—ELEPHANTIASIS OF THE LEGS

from malnutrition, the hairs may drop off and the nails become rough and thickened, while the skin perspires less and the sensation is diminished. Ulcers may now form on almost any part of the leg, especially about the knee,

If left untreated, the size of the leg gradually increases with repeated attacks of fever, and may reach considerable dimensions



FIG 720—ELEPHANTIASIS OF THE LEG BEFORE TREATMENT

in water) is given daily for three to six months the injections being interrupted for a few days from time to time. The injections may be made in the affected parts or deeply into the gluteal region

many patients cannot stand rubber bandaging. To increase the pressure on the hardest parts pads of inelastic material may be applied before bandaging and for this purpose small cylindrical

whole limb should be well distributed otherwise the parts on which insufficient pressure has been made will be found to become swollen. It is also useful to keep the affected limb continually elevated by means of pillows etc. In cases of verrucose elephantiasis in which the skin is covered with numerous horny masses a spirit lotion of resorcin and salicylic acid (ac salicylici resorcin aa gr xxx sp rect ziv) is useful in removing these horny masses.



FIG 721—THE SAME CASE AS IN FIG 720 AFTER TREATMENT BY FIBROLY IN AND BANDAGING AND WITHOUT ANY OPERATIVE MEASURES

the treatment gives much better and more lasting results in old-time

the diplococcus the blood and lymphangio-

In the cases so far reported there was marked improvement at first after operation but this did not last long. Madden and Ferguson report unfavourably on lymphangioplasty as a treatment for elephantiasis of the legs as they find the temporary improvement not maintained, because the reactive changes in the tissues immediately around the thrombi eventually obliterate the new vessels. Sittrunk reports favourably on the London Operation the aim of which is to establish by a wide excision of the aponeurosis a communication between the superficial and deep lymphatic channels.

Other methods of treatment consist in bandaging, massage, tapping with Southey tubes, and even amputation, but none of these are satisfactory.

#### Elephantiasis of the Scrotum.

The disease may begin as a lymph scrotum or with recurring erysipelatous attacks, with a red inflammatory blush on the skin, and fever. After each attack the scrotum is larger than it was before, and it goes on growing, if left alone, until it may reach the weight of 22½ pounds as mentioned by Chevers, which is probably the



FIG. 722.—ELEPHANTIASIS OF THE SCROTUM

are situated at the upper and back part of the tumour, and are usually surrounded by hydroceles.

If large, the base of the tumour is frequently ulcerated.



gauze bags filled with ordinary small lead shot are found especially useful. In some cases massage of the whole limb before bandaging is useful. It is of the utmost importance that the pressure on the whole limb should be well distributed otherwise the parts on which insufficient pressure has been made will be found to become swollen. It is also useful to keep the affected limb continually elevated by means of pillows etc. In cases of verrucose elephantiasis in which the skin is covered with numerous horny masses a spirit lotion of resorcin and salicylic acid (ac salicylici resorcin aa gr xxx sp rect ʒiv) is useful in removing these horny masses.



FIG 721—THE SAME CASE AS IN FIG 720 AFTER TREATMENT BY FIBROLYSIN AND BANDAGING AND WITHOUT ANY OPERATIVE MEASURES

In successful cases after three to six months of this treatment the affected parts are of much smaller size the skin is smoother more

mously thickened and melastic the coaptation of the opposed sur

is a slight and  
in the acute  
e other hand,

aponeurosis & communication between the superficial and deep lymphatic channels

Other methods of treatment consist in bandaging, massage, tapping with Southey tubes, and even amputation but none of these are satisfactory

#### Elephantiasis of the Scrotum.



FIG 722—ELEPHANTIASIS OF THE SCROTUM

**Treatment.**—The treatment is removal, which is a very easy operation, and very successful, following either Manson's or Charles's method. Manson says that the mortality need not exceed 5 per cent.

The most important feature of the whole operation is to have the skin perfectly clean and aseptic, and to carefully choose the parts of the skin which are to be used as flaps.

An elastic tourniquet in a figure of eight is applied round the pelvis and the neck of the tumour. The skin flaps are now marked out and deepened, the vessels being carefully ligatured as they are exposed.

The testicles are then dissected by perpendicular cuts, and the penis is set free by dissection after cutting down the canal already mentioned as formed by the prepuce. This is best done with a pair of scissors.

turned inside out, and after any redundancy has been removed, it is stitched around the testicle in the inverted condition. The flaps

be made for the penis, failing this, the raw area must be covered

operation an incision is made in the median line from near the pubis to the preputial mouth deepening it to the dorsum of the penis. Enucleation of the organ is performed from the suspensory ligament to its free extremity. There the glans is still separated from the

up, and the parts thoroughly cleaned. Now the mass is pulled to the patient's right exposing the left side of the neck of the tumour. Cut from at the median line with finger and thumb. The same procedure is repeated on the right side of the neck of the tumour. The two incisions will meet in front of the anus, all the main vessels will be seized and divided, and the bulb almost cleared on both sides.

Then the testicles are enucleated, and after wrapping them, as well as the cords, in gauze, they are placed on the pubis. The

flaps are now made practice determining the amount of covering necessary

Repeat the median line whilst a cc

Lastly flaps are stitched to the body of the penis

After the operation careful dressing well and equally applied ■ of great practical importance.

### Elephantiasis of the Vulva.

10 pounds or more : The treatment is removal

### Elephantiasis of the Breast.

Elephantiasis of the breast is very rare but does occur and the enlarged breast may reach to the pubes or the knee It may affect one or both breasts The treatment is removal

### Elephantiasis of the Arm

This is rare but may arise in the same manner as in the leg One arm or both arms may be affected Blair reports that the natives of Fiji and probably of other South Pacific Islands are in comparison with natives of other regions peculiarly liable to this form of elephantiasis

### Elephantiasis of the Scalp

Rarely the whole scalp is enormously thickened and presents deep furrows

### Circumscribed Elephantiasis

Large pendulous tumours of filarial origin one to several may be found These are commonest in the upper part of the thigh just below the groin

## RARER AFFECTIONS

Lewis has recorded a case of filariasis in which chyle containing *Microfilaria* was discharged from both conjunctivæ while Maitland has described cases of acute synovitis of the knee associated with filariasis as being of too frequent occurrence to be looked upon as merely coincidences



## CHAPTER LXVII

# THE MYIASES AND ALLIED CONDITIONS

The Myiases Rhinal, Aural, Ocular, Urinary, Vaginal, Oro gastro intestinal,  
Dermal—Allied conditions—References

### THE MYIASES.

**Definition.**—The myiases are the invasions of any part of the body of man or animals by dipterous larvæ

**Historical.**—The history of the myiases may be divided into 'ancient knowledge' and 'modern knowledge.'

*Ancient Knowledge*—As the disease is much more frequently

with the larva of the same fly. In the sixteenth century Ambrose

in the tropics.

These observations were extended in 1653 by Father Bernabé

tion—perhaps a myiasis—as occurring in soldiers

In 1687 Leuwenhoek, in Europe, mentions two cases, one of dermal myiasis in the leg of a woman who made a good recovery,

the 'ver

junior),

kind of mosquito, but from whence its egg comes is unknown. This observation has since been fully confirmed by recent research. In 1757 Arture drew attention to the occurrence of *Dermatobia hominis* in Cayenne, and in 1781 Linnæus junior did the same as regards Peru in a letter addressed to Pallas, in which he gave the fly its present name. Gmelin subsequently published this letter.

Somewhere about this time Turner described two cases of urinary myiasis in England.

In 1770 Wohlfart published an account of rhinal myiasis in his work entitled 'De Vermibus per nares excretis.'

In 1809 Azara gave a history of his journey in 1781-1801 into

in Jamaica, where it caused the death of a woman, and Sells gave an account of probably the same larva causing infestations of the

carpenter in Colombia, in 1835 Guyon mentioned a dermal myiasis in a negro in Martinique, in 1837 Hope described *Dermatobia hominis* in the head of a man, and called it *Æstrus guildingii*, after L. Guilding of Trinidad who found the case.

In 1840 there appeared the classical and much neglected work on the whole subject by Hope, in which not merely are the older accounts gathered together, but also clear definitions of the various conditions are provided.

From 1895 to the present time Austen has contributed many series of classical works by R. Blanchard on myiasis in general. From 1895 to the present time Austen has contributed many

in 1900, give a summary

**Ætiology.**—At the present moment too little is known as to the characters of the larvæ of the diptera to permit their recognition, unless belonging to a well known species, and it is obviously incorrect to assume that larvæ are those of a given fly. It is therefore necessary for the medical observer to —

- 1 Preserve specimens of the larvæ, as little damaged as possible, in 4 per cent formalin, and hold in position in the tube by means of fine tissue-paper
- 2 Rear the larvæ or pupæ and so obtain the imago, which should be fed for a day or so before being killed, and should then be carefully mounted and preserved. Mr. Austen has asked that some of the larvæ be preserved.



Cobo who states that in each wound caused by the common mosquito there grows a *spine covered worm* the size of a haricot bean or larger

About this time Fernellus described some form of nasal infestation—perhaps a myiasis—as occurring in soldiers

In 1687 Leuwenhoek in Europe mentions two cases one of

possible and from this time onwards the knowledge of the subject increased and improved In 1745 there appeared a work entitled

Relation abrégée d'un voyage fait dans l'intérieur de l'Amérique Méridionale by De la Condamine where on p 170 he mentions the ver macaque → e the larva of *Dermatobia hominis* (Linnæus junior) and says that it takes its birth in the wounds made by a kind of mosquito but from whence its egg comes is unknown This observation has since been fully confirmed by recent research In 1757 Arture drew attention to the occurrence of *Dermatobia hominis* in Cayenne and in 1781 Linnæus junior did the same as

reg

fly

myiasis in England

In 1770 Wohlfart published an account of rhinal myiasis in his work entitled *De Vermibus per nares excretis*

In 1809 Azara gave a history of his journey in 1781 1801 into ably

tion  
in Jamaica where it caused the death of a woman and Sells gave an account of probably the same larva causing infestations of the eyes ears nose and mouth in the same islands Some of Sells's cases ended in recovery and others in death Also about the same time (1806) comes the case of the soldier believed to be infested with *Cestrus hominis* Curtis in the skin near the scapula in Surinam

in

In

In 1840 there appeared the classical and much neglected work on the whole subject by Hope in which not merely are the older accounts gathered together but also clear definitions of the various conditions are provided

From the time of the publication of the first series of classical works by R. Blanchard on myiasis in general. From 1895 to the present time Austen has contributed many valuable articles dealing with these infestations. In 1903 Ward gave an excellent and well illustrated account of the larva of *Dermatobia*.

more necessary for the medical observer to —

1. Preserve specimens of the larvæ, as little damaged as possible, in 4 per cent formalin, and held in position in the tube by means of fine tissue paper.
2. Rear the larvæ or pupæ and so obtain the imago, which should be fed for a day or so before being killed, and should then be carefully mounted and preserved.

up in cigarette-paper, packed in small metal boxes and forwarded to England for identification.

3. Label all the specimens.
- 4.

principal families concerned are —

I *Muscidae* (Subfamily *Muscinae*) —Genera *Musca* *Calliphora*  
 Ch. . . . .

Chapter XXXIII p 814

Less important families are —*Tachinidae* *Micropezidae* *Syrphidae*  
*Phoridae* *Therevidae* *Sepsidae* and *Drosophilidae*

The larvæ appear to be attracted by faecal or urinary substances and also by any purulent or putrefactive discharges

**Pathology** —The changes produced in the body by these larvæ appear to depend upon the question of food. In such positions as the alimentary tract they appear to do little harm probably because there is plenty of food available without hurting the tissues of the host on the other hand in the nose ear and eye they may cause much destruction of tissue firstly by eating into the tissues secondly by the microbic infections which follow in their track and in this way they may cause the death of the host

**Symptomatology** —As may be expected from the last paragraph the symptoms of the victim may vary from nil local signs of destruction of tissue with inflammation and pus formation to signs of

in the alimentary canal by anthelmintic treatment when in the

follows —

A. *Internal or Cavity Myiasis* —

- I Rhinal myiasis
- II Aural myiasis
- III Ocular myiasis
- IV Urinary myiasis
- V Vaginal myiasis
- VI Gastro intestinal myiasis

## B External or Dermal Myiasis —

VII Traumatic dermal myiasis

VIII Subcutaneous myiasis

With regard to the ocular myiasis they may be a true cavity myiasis if the larva lives in the lachrymal sac but they may resemble a cutaneous myiasis if the larva penetrates into the tissues under the conjunctiva when indeed it may destroy the eye

## THE INTERNAL OR CAVITY MYIASSES

## RHINAL MYIASIS

For purposes of description this may be divided into the American rhinal myiasis the African rhinal myiasis the Asian rhinal myiasis and the European nasal myiasis

## American Rhinal Myiasis

## BICHEIRO

**Definition** — Bicheiro is a rhinal myiasis found in Tropical America and caused by the larvæ of *Chrysomyia macellaria* (Fabricius 1794) and allied species

**Climatology** — The causal fly extends really from Canada to Patagonia  
 justify  
 the colt  
 princip  
 Ætio

they lay eggs in the wounds of horses and mules produced by barbed wire in the sheaths of horses the vaginæ of mares and

in large numbers

In Trinidad Lawrence reports that the disease may be caused by *Chrysomyia viridula* Robineau Desvoidy

**Pathology** — The eggs deposited in the manner just described become larvæ in the course of a couple of days The larvæ proceed to feed upon the tissues of the nose and to burrow deeply into the mucous membrane down to and even through the bone The

of the nose, pharynx, hard and soft palates, larynx, etc. Fistulous channels may be seen packed with larvæ and extending in all directions.

**Symptomatology.**—Some couple of days after a person suffering from a chronic catarrh, foul breath, or ozæna, has slept in the open, or has been attacked by a fly when riding or driving—i. e. when the

presence of the larvæ. Left untreated, the patient rapidly becomes worse, and pus and blood are discharged from the nose, from which an offensive odour issues. Cough appears as well as fever, and often some delirium. If the patient lives long enough the septum of the nose may fall in, the soft and hard palates may be



FIG 723—*Chrysomya macellaria* LARVA (X4) (After Blanchard)

pierced, the wall of the pharynx may be eaten away, exposing the vertebrae and even the hyoid bone may be destroyed. By this time, however, the course of the disease will have become quite evident by the larvæ dropping out of the nose and if the patient continues to live all the larvæ may come away naturally.

**Diagnosis.**—Any case with the history of a fly having darted into the nose should be assumed to be a case of nasal myiasis until proved by careful nasal examination not to be so. The onset of peculiar sensations at the root of the nose and along the orbital processes together with the signs of an aggravated nasal catarrh

Calomel may be insufflated after douching. It may be necessary to open the frontal or other sinuses and to irrigate the passages.

**Prophylaxis**—The prophylaxis consists in the avoidance of sleeping in the open air except under a mosquito curtain and in the prompt treatment of any case in which a fly has been known to enter the nose.

### The African Rhinal Myiasis

THIM NI

**Oriental** It also occurs in the Ahaggar Mountains of the Central Sahara.

**Ætiology**—*Oestrus ovis* (pp. 826, 827) should lay its eggs in the

injections or washes but in the eye gentle removal is all that is required.

### OTHER AFRICAN RHINAL MYIASSES

In addition to thim ni rhinal myiasis in Africa is known to be due to—*Lucilia hominivorax* (vide Bouchet at Barika in 1895)

*Sarcophaga nura* Rudolphi, by Mouchet at Katanga, *Pycnosoma bitotum* Wiedemann, 1830, in Abyssinia Belgian Congo, and Lorenzo Marques. The condition has been reproduced experimentally in animals by Wellman, in 1906, with *S. regularis* Wiedemann in a goat.

### Asian Rhinal Myiases.

#### PEENASH

**Definition.**—Peenash is a word which may be used for the Indian rhinal myiases caused by the larvæ of species of *Pycnosoma* Brauer and von Bergenstamm and by larvæ of species of *Sarcophaga* Meigen, especially by those of *Sarcophaga carnaria* Linnæus 1758.

**Treatment.**—This is the same as for other forms (*vide supra*).

### European Rhinal Myiases.

These are known to be caused by *Piophilæ casei* and by species of *Sarcophaga* Meigen 1826 while *Calliphora limea* is also said to be causal.

#### AURAL MYIASIS

**Definition.**—Aural myiasis is the invasion of the external auditory meatus, the middle ear, and associated cavities by the larvæ of certain dipterous flies, especially those of the Muscidae and Sarcophagidae.

**Historical.**—Aural myiases have been recorded by Taschenberg in 1870, Blake in 1872, Johnson in 1892, and Austen in 1912, but the most complete study is that by Francavilla in 1914.

**Ætiology.**—The following larvæ have been noted as causal agents—

*Muscidae* (Subfamily Muscinæ) —

*Musca domestica* Linnæus

1794

*Sarcophagidae* —

*Sarcophaga carnaria* Linnæus 1758, synonym, *S. carnosa* L., 1758

*Sarcophaga magnifica* Schiner, 1862, synonyms *S. uohlfarti* Portschinsky, 1875, *S. ruralis* Meigen, *S. meigeni* Portschinsky.

*Anthomyiidae* —*Tannia scalaris* Meigen*Fannia canicularis* Linnæus*Fannia incisurata* Zett*Hydrotaea meteorica* Linnæus*Syrphidae* —*Syrphus* sp<sup>?</sup>*Æstridae* —*Æstrus ovis* Linnæus 1761

**Symptomatology** — If lodged in the external auditory meatus they may simply cause deafness and ringing in the ears but if eating into the middle ear they may give rise to a discharge of blood and pus

**Prophylaxis** — Some protection such as wool is necessary when suffering from an aural discharge

## OCULAR MYIASIS

**Definition** — Ocular myiasis is the invasion of the spaces under the eyelids the lachrymal sac the subconjunctival tissue or the

of a species of hypoderma in the anterior chamber of the eye of a girl

**Ætiology** — The larvæ so far recognized as causing this form of myiasis are —

*Sarcophagidae* —*Wohlfartia magnifica* (Schiner 1862)*Necrobia* sp<sup>?</sup>*Æstridae* —*Æstrus ovis* Linnæus 1761*Dermatobia cyaniventris* Macquart 1843*Hypoderma* sp<sup>?</sup>

**Symptomatology** — This varies from the discovery of a larva lying like a foreign body under the eyelid to infection of the lachrymal sacs or the tissue under the palpebral or ocular conjunctiva to the total destruction of the eyeball

**Treatment** — This consists of the prompt removal of the larva by surgical means



**Prophylaxis**—The prophylaxis consists of prompt treatment of conjunctivitis and the protection of the head when sleeping out of doors. Natives wrap themselves up completely when sleeping out of doors.

#### URINARY MYIASIS.

**Historical.**—This form of myiasis is rare, but has been recorded by Ambrose Paré in 1582 and by Turner in the seventeenth century, when he recorded two cases, while in 1909 René Chervel analyzed all cases reported up to that date, and concluded that, of twenty reported cases, six were genuine, ten were probable, and four were doubtful. He also added one of his own observation. King, in 1914, reported an American case. Palmer and Austen have recorded a case in England. Hagen has also drawn attention to a case in Boston.

**Ætiology.**—It is thought that the flies deposit the eggs near the meatus urinarius and that the newly born larvæ pass up into the urethra. Sleeping in the open is generally accused as the method of infection, but paralyzed persons become infected, especially those with urinary troubles. The larvæ which so far have been recognized are those of *Fannia canicularis* Linnæus, 1761, and *Fannia scalaris* Fabricius (p. 852).

**Symptomatology.**—Generally the larvæ are discovered accidentally when passing urine, when they may cause some slight obstruction.

**Treatment.**—This apparently is unnecessary, as the larvæ come away without causing harm.

#### VAGINAL MYIASIS.

**Definition.**—Vaginal myiasis is the invasion of the vagina by the larvæ of dipterous insects, especially those of the Muscidae.

The vagina of a beggar woman and Low has seen a similar case in the West Indies.

#### ORO-GASTRO-INTESTINAL MYIASIS.

**Definition.**—Intestinal myiasis is the invasion of the intestine by the larvæ of certain species of flies, but especially by those belonging

to the genera *Sarcophaga* (p 830), *Fannia* (p 852), and *Aphiocha* (p 824)

History.—Many stray cases of myiasis—e g Jenyns (1839)—have been reported from time to time, but these have been gather

Garrod and by Soltan in the *Journal of Parasitology*, 1910. They have seen several cases in Ceylon and in the Balkans. Cases have been reported in England by Stephens (1905), Hewitt (1906), Cattle (1906), Garrod (1909), Soltan (1910) and Austen (1912)

Ætiology.—Intestinal myiasis may be produced by the larvæ of the following species —

## SARCOPHRAGIDÆ —

- Sarcophaga carnaria* Linnæus 1758
- hemorrhoidalis* Fallen 1810
- hamatodes* Joseph
- affinis*
- magnifica* Schiner 1862
- wolffarti* Portschinsky 1875
- laticornis* Meigen
- ruralis* Fallen
- meigeni* Schiner
- Cynomyia mortuorum* Linnæus 1761

## ANTHOMYIDÆ —

- Fannia canicularis* Linnæus 1761
- incituralis* Lett
- manicula* Meigen
- salatrix*
- deyardensis*

*Hydrotaea meteorica* Linnæus

## MUSCIDÆ —

- Musca domestica* Linnæus
- corvina* Fabricius
- nigra*
- Cyrtoneura stabulans* Macquart
- Pollenia rudis* Fabricius
- Calliphora vomitoria* Linnæus 1758
- erythrocephala* Meigen
- arvea*
- Lucilia caesar* Linnæus
- vegana* Macy
- Chrysomya putida* Linnæus
- Tachomyia fusca* Macquart (?)

## TACHINIDÆ —

- Tachina larvarum* Meigen

## MICROPHYIDÆ —

- Calobata esbiana* Meigen

## SYRPHIDÆ —

- Eristalis tenax* Fabricius  
 „ *arbustorum* Fabricius  
 „ *dimidiatus*  
*Helophilus pendulinus* Meigen

## PHORIDÆ —

- Aphiochata ferruginea* Brunner  
*Phora rufipes* Meigen, synonym *P pallipes* Latreille

## THEREVIDÆ —

- Thereva nobilitata*

## SEPSIDÆ —

- Piophilus cases* Linnæus

## DROSOPHILIDÆ —

- Drosophila funebris* Meigen  
 „ *melanogastra* Brunner

## CÆSTRIDÆ —

- Spilogaster divisa* Meigen  
*Gastrophilus pecorum*

Sometimes more than one species may be found in the same case. Rarer forms of myiasis are those by larvæ of the Tipulidæ, by the bots of *Gastrophilus equi* mentioned flies enter the ally with vegetable food in an cooked or partially cooled condition. Another method is for fly to deposit its eggs on the nostrils and lips of children from which they pass into the stomach and intestines, and a third method entry of the larvæ into the rectum while using a privy. Intestinal myiasis is not uncommon in cattle, both in the Temperate one and in the tropics. There seems to be no doubt that the rvæ can live for a considerable time in the intestine but the most arked example of this is *Aphiochata ferruginea* Brun, which is believed to be capable of passing through its entire life-cycle in the human colon because both newly hatched and fully grown rvæ were passed by a patient every two months for nearly a year,

ere found in numbers in a corpse exhumed at La Fayette, Indiana, S A, two years after burial

If there is any truth in this statement, it proves that the life cycle of these flies can be completed in a parasitic state, and would explain the possibility of a patient suffering from myiasis for twelve years, and during treatment passing 1,000 to 1,500 larvæ. Enwick also reports cases in which it seemed probable that the whole life cycle was completed in man. It is obvious that these statements require careful confirmation by other similar cases

**Morbid Anatomy.**—We are not acquainted with the details of any post mortem examination in man, but in dogs the mucosa of the stomach and marked by numerous small hæmorrhages enter the blood through

the period varying from four to six days. The larvæ has a period of 10 to 14 days, first, second, third, fourth, fifth, sixth, seventh, eighth, ninth, tenth, eleventh, twelfth, thirteenth, fourteenth, fifteenth, sixteenth, seventeenth, eighteenth, nineteenth, twentieth, twenty-first, twenty-second, twenty-third, twenty-fourth, twenty-fifth, twenty-sixth, twenty-seventh, twenty-eighth, twenty-ninth, thirtieth, thirty-first, thirty-second, thirty-third, thirty-fourth, thirty-fifth, thirty-sixth, thirty-seventh, thirty-eighth, thirty-ninth, fortieth, forty-first, forty-second, forty-third, forty-fourth, forty-fifth, forty-sixth, forty-seventh, forty-eighth, forty-ninth, fiftieth, fifty-first, fifty-second, fifty-third, fifty-fourth, fifty-fifth, fifty-sixth, fifty-seventh, fifty-eighth, fifty-ninth, sixtieth, sixty-first, sixty-second, sixty-third, sixty-fourth, sixty-fifth, sixty-sixth, sixty-seventh, sixty-eighth, sixty-ninth, seventieth, seventy-first, seventy-second, seventy-third, seventy-fourth, seventy-fifth, seventy-sixth, seventy-seventh, seventy-eighth, seventy-ninth, eightieth, eighty-first, eighty-second, eighty-third, eighty-fourth, eighty-fifth, eighty-sixth, eighty-seventh, eighty-eighth, eighty-ninth, ninetieth, one hundred, one hundred and one, one hundred and two, one hundred and three, one hundred and four, one hundred and five, one hundred and six, one hundred and seven, one hundred and eight, one hundred and nine, one hundred and ten, one hundred and eleven, one hundred and twelve, one hundred and thirteen, one hundred and fourteen, one hundred and fifteen, one hundred and sixteen, one hundred and seventeen, one hundred and eighteen, one hundred and nineteen, one hundred and twenty, one hundred and twenty-one, one hundred and twenty-two, one hundred and twenty-three, one hundred and twenty-four, one hundred and twenty-five, one hundred and twenty-six, one hundred and twenty-seven, one hundred and twenty-eight, one hundred and twenty-nine, one hundred and thirty, one hundred and thirty-one, one hundred and thirty-two, one hundred and thirty-three, one hundred and thirty-four, one hundred and thirty-five, one hundred and thirty-six, one hundred and thirty-seven, one hundred and thirty-eight, one hundred and thirty-nine, one hundred and forty, one hundred and forty-one, one hundred and forty-two, one hundred and forty-three, one hundred and forty-four, one hundred and forty-five, one hundred and forty-six, one hundred and forty-seven, one hundred and forty-eight, one hundred and forty-nine, one hundred and fifty, one hundred and fifty-one, one hundred and fifty-two, one hundred and fifty-three, one hundred and fifty-four, one hundred and fifty-five, one hundred and fifty-six, one hundred and fifty-seven, one hundred and fifty-eight, one hundred and fifty-nine, one hundred and sixty, one hundred and sixty-one, one hundred and sixty-two, one hundred and sixty-three, one hundred and sixty-four, one hundred and sixty-five, one hundred and sixty-six, one hundred and sixty-seven, one hundred and sixty-eight, one hundred and sixty-nine, one hundred and seventy, one hundred and seventy-one, one hundred and seventy-two, one hundred and seventy-three, one hundred and seventy-four, one hundred and seventy-five, one hundred and seventy-six, one hundred and seventy-seven, one hundred and seventy-eight, one hundred and seventy-nine, one hundred and eighty, one hundred and eighty-one, one hundred and eighty-two, one hundred and eighty-three, one hundred and eighty-four, one hundred and eighty-five, one hundred and eighty-six, one hundred and eighty-seven, one hundred and eighty-eight, one hundred and eighty-nine, one hundred and ninety, one hundred and ninety-one, one hundred and ninety-two, one hundred and ninety-three, one hundred and ninety-four, one hundred and ninety-five, one hundred and ninety-six, one hundred and ninety-seven, one hundred and ninety-eight, one hundred and ninety-nine, two hundred.

able. Pain is very severe and vomiting may occur, and sometimes hæmatemesis, while the larvæ attain

repeated examinations, the nature of the larvæ in nasal myiasis usually come from the

with a dose of castor oil should be adminis-

the face should be washed and by the use of salads should be

avoided

### Ovanya.

Wellman, associated with *gardensis*

### Muculo.

This is the African myiasis, concerning which the editors of the *Journal of Tropical Medicine and Hygiene* asked for information in 1907, but which so far has not been traced

## THE EXTERNAL OR DERMAL MYIASSES

These are the infections of the skin, whether wounded or not by dipterous larvæ. There are two varieties of this type—viz., traumatic dermal myiasis and subcutaneous myiasis.

### Traumatic Dermal Myiasis.

**Definition.**—Traumatic dermal myiasis is the invasion of wounds or ulcers of the skin by the larvæ of dipterous insects, principally belonging to the Muscidae and Sarcophagidae

**Historical.**—One of the earliest publications with references to this is Joseph in his 'Myiasis Externa Dermatosa,' published in Hamburg in 1800, but a large number of observations have been published since then

**Ætiology.**—The larvæ which have been recognized so far are —

*Muscidae* (Subfamily Muscinae) —

*Chrysomya macellaria* Fabricius

*Chrysomya viridula* Robineau Desvoidy

*Calliphora*, sp. ?

*Lucilia argyrocephala* Macquart

*Lucilia*, sp. ?

*Cordylobia anthropophaga*

*Musca putrida*

*Sarcophagidae* —

*Sarcophaga carnaria* Linnæus, 1758

„ *magnifica* Schiner, 1862

„ *ruficornis*

„ *chrysoloma* Wiedemann

„ *plinthopegga* Wiedemann (The adult is one of the 'yaws flies' of Dominica)

„ sp. ?

**Symptomatology.**—The larvæ accentuate the putrid condition of the sores and the sufferings of the patient

**Treatment.**—Antiseptic douches syringing, with removal of the larvæ, and subsequent antiseptic dressing

**Prophylaxis** —The myiasis can, of course, be prevented by simply applying aseptic dressings to wounds

### Subcutaneous Myiasis.

**Synonyms** —Cutaneous myiasis *French* Myase cutanée, Myase furon culeuse *Myase rampante sous cutanée*, *Italian*, Myasis cutanea, *German* Myasis

**Definition** —Dermal myiasis is the invasion of the skin by the larvæ of species of the *Æstridæ*, especially by *Dermatobia cyaniventris* Macquart, 1843, and by those of the *Muscidae*, especially *Cordylobia anthropophaga* Blanchard

of the fly causing the myiasis, and therefore this should be bred out as described in the opening sections of this chapter

**Ætiology.**—The following larvæ are known to cause subcutaneous myiasis in man —

*Cestridæ* —

*Hypoderma bovis* de Geer

*Hypoderma lineata* de Villiers

*Hypoderma diana* Brauer

*Dermatobia cyaniventris* Macquart, 1843, synonym, *D. hominis* (Linnæus junior, 1781)

*Dermatobia* (?) *kenia* Kolb

*Estrus* (*Cephalomyia*) *ovis* Linnæus

*Muscidæ* (Subfamily Calliphorinæ) —

*Cordylobia anthropophaga* E Blanchard

*Cordylobia rodhaini*

The life history of the cestridæ is curious, as will be discussed

American, the African, the Asian, and the European, while the last variety is not geographical, but pathological—viz, creeping eruption

**American Dermal Myiasis.**

**Synonym.**—Neotropical dermal myiasis, Dermatobiasis.

has a large

junior, 1781),

**Popular Names**—Brazil, húra (boil), verme, berne or berme, British Honduras, beef worm, cormollote, Colombia and Venezuela, gusáno, husano (worm), gusáno de monte (forest worm), gusáno peludo (hirsute worm), gusáno macaco (macaw worm, because it attacks the macaw headed Capuchin monkeys), Costa Rica, torcel, Guatemala, colmoyote, Pangoa, mirunta, Mayan name, suglacura

**Names Suggestive of Mosquito Carriage**—Mexico, moyocuil

History.—In 1569 Friar Pedro Simon appears to have been the

in the state of Vera Cruz In 1745 De la Condamine reported it from French Guiana, as did Arture in 1757 In 1781 Linnaeus junior reported it from Peru and gave a brief mention of the fly while in 1822 Say gave a description of the larva as received from South America In 1835 Hope gave an account of the larva from under the skin of the head of a man from Trinidad The specimen was deposited in the museum of the Royal College of Surgeons of England

From that date scattered but fairly numerous, references can

observations

With reference to the mosquito carriage it is remarkable to note that Father Cobo in 1653 says that each wound produced by the common mosquito produces *within the flesh a spine covered with the size of a haricot bean or even larger*

In 1911 Morales of Guatemala first described the transmission

puration removing the larva transplanting it into the back of a rabbit and watching its escape as a nymph Also in 1911 Tovar of Maturin in Venezuela had noted this mosquito carriage, which

near the southern borders of  
Mexico in Central America  
Guatemala and Costa Rica  
— i. e., Colorado

insect the  
a in which  
valleys of  
It begins  
United States being found in  
h and Spanish Honduras  
Panama in South America  
Brazil and Peru, while

it is known to occur among the West Indian Islands near South America—e g , Trinidad

It requires a warm, moist climate with surface water and forest vegetation

Ætiology.—The cause of the disease is the invasion of the body by means of the larva of *Dermatobia hominis* (Linnæus junior 1781).

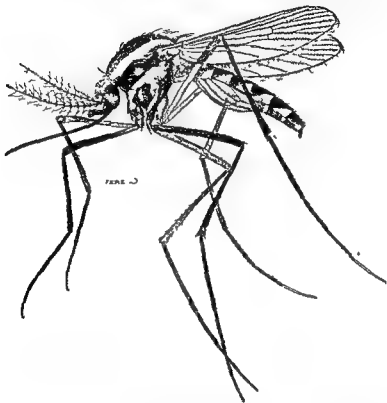


FIG 724 —*Janthinosoma lutzi* THEOBALD CARRYING THE EGGS OF *Dermatobia hominis* LINNÆUS JUNIOR  
(After Sambon)

On the evidence at present available it would seem that the fly seizes the female mosquito of the species *Janthinosoma lutzi* Theobald



and pierces the skin by means of the aperture made by the mosquito bite

**Pathology**—As it escapes from the egg the larva possesses in addition to the numerous spines on its first seven segments a crown of large black rose thorn shaped spines along the anterior border of the fifth sixth and seventh segments while the last shows two posterior stigmata. When it has pierced the skin the spines on the



FIG 725—WARBLE CAUSED BY *Der  
matobia hominis* (LINNÆUS JUNIOR)  
(After Sambon)

and the pain will increase as the little rounded swelling which appears in the affected region increases in size. This swelling is the warble.

**The Warble Stage**—When fully developed the warble resembles a boil being some 2.3 centimetres in diameter and of a dark red or bluish red colour. At the apex there is a more or less centrally placed small circular aperture which

increases till it reaches a size of 3.6 millimetres. It is usually covered by a scab which if removed shows a moving body with two small brownish yellow spots. This is the posterior end of the larva. These warbles may be single or multiple placed in close proximity or scattered with usually only one larva to a warble but sometimes with more and rarely as many as five larvæ to one warble. Warbles may exist in any part of the body but are more painful in regions like the nose. Usually there are no constitutional symptoms but there may be slight fever and there may be swelling in the head when the adhesions of the warble may be seen appearing and disappearing from the aperture. In due course it gradually dilates the opening in the warble by means of its posterior end and eventually escapes and falling to the ground crawls away and becomes a pupa.

*Post-Warble Stage*—After a period varying from six weeks to six months the larva escapes and leaves behind an empty cavity, which is closed by granulation tissue and heals, leaving a hardly visible scar.

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### African Dermal Myiasis.

The known form of African dermal myiasis is *tumbu disease* caused by members of the *Calliphorinae* subfamily of the *Muscidae*.

#### TUMBU DISEASE

*Definition*.—Tumbu disease is an African dermal myiasis caused

1905

In 1901 it was reported that there was a maggot fly in Natal, limited to the coast, not extending inland and not rising higher than 1,000 feet. It was also found about Delagoa Bay, in Rhodesia, British Central Africa, Uganda, and the Anglo Egyptian Sudan.

the whole subject in 1908 to the *Journal of the Royal Army Medical Corps*, while Smith described the symptoms of the disease in the same journal

In 1913 Rodham, Pons, Vandenbranden, and Bequaert, demonstrated by experiment the method of infection, and Heckenroth and Blanchard recorded four cases due to *Cordylobia rodhami* in French Equatorial Africa

**Ætiology.**—The cause of the disease is the invasion of the subcutaneous tissues by the larva of *Cordylobia anthropophaga* and allied species. The method of infection thanks to the labours of Smith in 1908, and Rodham, Pons, Vandenbranden, and Bequaert in 1913 is fairly well known

and buttocks in Europeans but the forearm (especially in natives) and the head also may be invaded. The infected area presents the appearance of a boil in the central portion of which there is an opening more or less clearly defined which is marked by black matter (the excrement of the larva). In this hole the posterior end of the larva may be noted and on pressing on it considerable pain is produced probably due to the movements of the larva. Surrounding the central opening is an inflamed area about  $\frac{1}{2}$  inch in diameter. The opening may however be obscured by dried up discharge. Strong pressure easily expels the maggot, children and helpless usually associated 12 millimetres in jointed anteriorly, the

and the wound dressed aseptically

### Asian Dermal Myiasis.

Myiasis has not been sufficiently studied in Asia though it is fairly common there. *Sarcophaga ruficornis* is reported as causing occasionally a very severe form of cutaneous myiasis but beyond this much cannot be said.

### European Dermal Myiasis.

The myiasis of Europe scarcely comes under the scope of tropical medicine but it may be mentioned that dermal myiasis are known to be caused by *Hypoderma bovis* de Geer 1776 (p. 826) *H. diana* Brauer *H. lineata* de Villiers 1789 *Gastrophilus nasalis* Linnæus 1758 *G. hemorrhoidalis* Leach 1761 *G. veterinus* and *Lucilia sericata*.

### Creeping Eruption

**Synonyms**—Creeping disease Larva migrans Bulgarian Nova Bolest, Ixasta Bolest German Hautmilwaf Kreechkrankheit Hautkratzchorf

**History**—This disease was first described by A. Lec in 1875. Later on Procke Blanchard Topsent Gulleborn and others have recorded several cases. It is not rare in some parts of Europe Africa and America. It is numerous

*strophilus*  
*Sarcophaga ruficornis* *Gastrophilus* *strophilus* *Hypoderma bovis* and *H. lineata* have been found in several cases. In others no larva whatever was found. Icoos states that the same clinical picture may be caused occasionally by *Ancylostoma* and *Strongyloides (Anguillula)* larvæ or even by an inanimate object like a piece of horsehair. A larva has been found 2 millimetres from the inflamed end under a small dark spot.

**Symptomatology**—The eruption is characterized by the presence of a narrow raised red line  $\frac{1}{4}$  to 1 inch broad. This line extends daily one or several inches and is generally sinuous but may be straight. While the advancing end progresses the opposite end slowly fades away. The duration of the malady is long generally several months but occasionally two or three years. There is much pruritus.

**Treatment**—Hypodermic injections of various disinfectants have been tried with little success. Hutchins recommends a cocaine injection followed by the injection of 1 or 2 drops of chloroform.

### ALLIED CONDITIONS

Allied to the myiasis are infestations by the larvæ or imagines of the Lepidoptera Coleoptera Diplopoda Chilopoda and Dermoptera.

## Scolechiasis

**Synonym** —Scholechiasis

**Definition** —Scolechiasis is the invasion of the body by the larvæ of the Lepidoptera

**History** —Originally the term 'scolechiasis' was used by Kirby and Spence for the invasion of the body by the larvæ of any insects but Hope suggested that the term should be restricted as indicated and invented new terms for other infestations

**Varieties** —Hope gives a list of seven cases of which five were gastro intestinal one was rhinal and one was not classified

**Gastro Intestinal Scolechiasis** —This is due to *Pontia brassica* Linnæus belonging to the Papilionidæ and observed by Calderwood in Scotland Two were due to *Crambus pinguinalis* of the Noctuidæ one being observed by Linnæus in Sweden and one by Church in England and one was due to *Phryganea grandis* observed by Church in England The one without determination was found by Lister in England

**Rhinal Scolechiasis** —There is only one case caused by *Crambus pinguinalis* and recorded by Kirby and Spence on the authority of Fulvius Angelinus as occurring in Ravenna

## Canthariasis

**Synonym** —Scolechiasis as used by us in the second edition

**Definition** —Canthariasis is the invasion of the body by the larvæ of the Coleoptera

**Remarks** —The term used above was introduced by Hope in 1840 when he recorded a number of cases of rhinal gastro-intestinal and urinary infestations

**RHINAL CANTHARIASIS** —This was due to *Tenebrio moletor* Linnæus and was recorded by Tulpius and by Oswald Allen

**GASTRO INTESTINAL CANTHARIASIS** —The genera recorded are numerous

**Carabidæ** —*Sphodrus leucophthalmus* by Paykull in Sweden in 1797 *Dermestes lardarius* by Otto and Chuchestei in 1807 in

*orus subter*

*S politus*

Fabricius *S fuscipes* Fabricius and *S punctulatus* Fabricius by Paykull in Sweden in 1796 1798

**Scarabæidæ** —*Geotrufes icinalis* by Van Brommell in Sweden in 1729 *Melolontha* sp ? by Lemaout Depalse (1817?) and Desvoidy in France

**Tenebrionidæ** —*Tenebrio moletor* Linnæus by Forestus in 1568 at Brielle by Kelle in Scotland by Pickells and by Thomson in Ireland by Traull and others in England by Acrel in Sweden in 1796

**Blapidæ** —*Blaps mortisaga* Fabricius by Pickells Thomson and O'Brien in Ireland in 1827 by Bateman and others in England

*Mordellidæ* — *Mordella*, sp ? by Rosen in 1752 in Sweden

*Cantharidæ* — *Melæ proscarabæus* Fabricius by Germar in Silesia in 1816

*Circulionidæ* — *Balaninus nucum* Fabricius by Henry, Astley Cooper and others in England in 1805 1809

URINARY CANTHARIASIS — *Tenebrio moletor* Linnæus is recorded by Tulpinus as occurring in the bladder of a female aged fifty years  
*Balaninus* sp ? by Henry and Phillips in the urinary passages of a man aged sixty two years in Lancashire in 1809 King in the

### Diplopodiasis and Chilopodiasis

*Diplopodiasis* occurs in the alimentary canal and is caused by *Julus terrestris* Linnæus *J. londonensis* and *Polydesmus complanatus*

*Chilopodiasis* occurs in the rhinal passages where *Geophilus carpophagus* Leach *G. electricus* Linnæus *G. cephalicus* Wood *G. similis* Leach *Lithobius fortificatus* Linnæus and *L. melanops* have been found while *G. electricus* and *S. coleoptrata* Chatechelyne *vesuviensis* *Himantarium gervaisi* and *Stigmatogaster subterraneus* occur in the alimentary canal

### Dermapteriasis

*Dermapteriasis* of the alimentary canal caused by *Forficula auricularia* has been reported by Griffin in 1836

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## CHAPTER LXVIII

# POROCEPHALOSIS

Synonym — Definition — History — Climatology — Ætology — Pathology —  
Morbid anatomy — Symptomatology — Diagnosis — Prognosis — Treat-  
ment — Prophylaxis — References

**Synonym** — Porocephalasis

**Definition** — Porocephalosis is the invasion of the body by the larvæ of *Porocephalus armillatus* Wyman 1848 and *P. moniliformis* Diesing 1836 and possibly other forms which become encysted in the liver and lungs and ultimately develop into nymphæ which may by their wanderings cause inflammation of organs and serous membranes

**History** — *P. armillatus* was first found in man by Pruner in 1846 and has since been studied by Bilharz Tenger Aitken Giard Broden and Rodhain Dutton and Todd and one of us *P. moniliformis* has been met with in Ama



FIG 726 — *Porocephalus armillatus*  
ENCYSTED IN LIVER

(After Sambon from our West  
African case)

and by Salm in Java Sambon thinks that Welch's Indian parasite may have been *P. naja* Leuckart 1860 or *P. crocidura* Parona 1890 found in Blyth's musk shrew *Crocidura fuliginosa* and that Flint's case in America may have been an infection with *P. crotali* Humboldt 1808 but for details with regard to these parasites see pp 734 and 736

**Climatology** — *Porocephalus armillatus* is confined to Africa being met with in negroes resident or who have resided

therein It has been reported from Egypt and various parts of

might well be a *Porocephalus*

**Ætiology** — The adults of both *P. armillatus* and *P. moniliformis* live in the nasal cavities and lungs of pythons and snakes and though the life-history is as yet unknown it is quite possible that

the victim

**Morbid Anatomy**—In opening the abdomen the nymphæ may

and lungs

In the cysts they lie in a curved position with the ventral surface on the outer aspect of the curve. The lungs show signs of bronchitis and pneumonia and the peritoneum is usually chronically inflamed but not always.

**Symptomatology**—The symptoms of the early stages of the disease are at present quite unknown but the terminal symptoms are emaciation and weakness associated with attacks of bronchitis, pleurisy or other respiratory symptoms. There may be cavities in the lungs and the sputum may be offensive and may contain the parasites of which as many as 75 to 100 have been recorded as being expectorated by a single patient. The liver is usually considerably enlarged.

**Diagnosis**—The disease has often been mistaken for phthisis therefore any patient in the tropics suffering from the usual symptoms of phthisis associated with enlargement of the liver may be suspected and the sputum and fæces carefully watched for the possible appearance of the parasites. When a parasite is found it may not necessarily be either *P. armillatus* or *I. moniliformis* but is more likely to be some form found in some animal which lives in the region where the patient resides or works.

**Prognosis**—The disease is generally chronic. The prognosis is serious.

**Treatment**—There is no known treatment.

**Prophylaxis**—If the drinking water is boiled or filtered there ought to be no danger of infection.

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## CHAPTER LXIX

### LEPROSY

Synonyms—Definition—History—Climatology—Ætiology—Symptomatology  
—Diagnosis—Prognosis—Treatment—Prophylaxis—References

Synonyms—*Elephantiasis Græcorum* French *La Lèpre* Italian *Lebbra*  
German *Aussatz* Norwegian *Spedalskhed* Arabic *Djudsam*

**Definition**—Leprosy is a chronic general disease caused by the *Mycobacterium lepræ* Hansen 1874 (usual term *Bacillus lepræ*) which produces characteristic lesions in the skin mucosæ and nerves. The method of infection is unknown.

**History**—Even at the present time there is occasionally much difficulty with regard to the diagnosis of leprosy from allied diseases and therefore in ancient times and in the Middle Ages syphilis and skin diseases without doubt were confused with it. Hence the history is not easy to write. Nevertheless such a repulsive and striking disease must have been noticed by the ancients and therefore it is possible that the references in the Ebers Papyrus and in the English Bible the Rig Veda and ancient Japanese books actually refer to what we call leprosy together with other diseases. If this is so leprosy is indeed ancient and wide-spread. Certain authorities however are of opinion that the Hebrew word Tsaraath which the translators of the Bible have rendered as leprosy does not refer to such disease. It is probable that the malady passed from Egypt to Greece and later to Italy by means of Pompey's troops and that it was disseminated throughout Europe by the Roman legions by traders and later perhaps by the Crusaders returning from the East. In an early

was diag  
The result  
of lepers

diminished rapidly in the fourteenth and fifteenth centuries since when the disease has almost disappeared from many parts of Europe

But while abating in Europe it appears to have been introduced into Madeira and the Canary Islands about the end of the fifteenth century and perhaps also into America by the Spaniards The

while it is very common in South China During recent years the Chinese have been moving about the world and are accredited with introducing the disease into Kamschatka the Sandwich Islands Polynesia Columbia California Australia New Zealand and also into Indo China

Though very common in North and Tropical Africa it appears to have been unknown in South Africa until introduced in 1756 by the Dutch from Java who carried it through Cape Colony and the Orange Free State into the Transvaal It must however be stated that some people think it has existed for a long time in South Africa

It is said that the numbers of lepers have markedly increased in South Africa since the advent of the East Indian troops in the middle of last century

As regards medical literature Hippocrates says but little about the disease and perhaps really refers to psoriasis while Aristotle defines it better It is not however until the first century that Aretæus of Cappadocia gives 1847 that the first Boeck appeared studied by Virchow baco Innes Camp

probably no connection between the human and the rat disease

various theories more or less ably defended which will be mentioned later Marchoux and Bourret consider that they have successfully inoculated a chimpanzee and Nicolle and Blazot have

importantly by Borthen in 1899

Deycke and Reschad inoculated the surface cream of sterilized unskimmed milk with material obtained from the under aspect of leprotic tubercles by throwing back a flap of skin Incubated is characterized by the presence of a substance called *Streptothrix nastin* which is similar to a fat found in Hansen's bacillus and this Deycke considered to be the agent which produced favourable symptoms

which it deprives of their fat and so allows the phagocytes to attack them Four solutions were prepared NASTIN B 0 NASTIN B 1 NASTIN B 2 and K this last being only benzoyl chloride and being used to shorten and reduce the severity of the reaction if required The others represent nastin in varying degrees of strength NASTIN B 2 containing an excess of nastin while NASTIN B 1 is that usually employed Unfortunately no general success has followed this line of treatment

There is but little leprosy in Iceland while it is common in Crete and Cyprus Sweden Greece and some of the Mediterranean islands rare in France and Germany and almost extinct in Denmark Belgium Holland Austria Hungary and England

It is very common throughout the whole of Asia In Ceylon there are numerous lepers many of whom are treated in a leper asylum at Hendela near Colombo which is believed to be one of

the Sandwich Islands and New Zealand it is common in Tahiti

and the Sandwich Islands.

It is spread sporadically over the United States but is rare in Canada while it is well known in Mexico and Central America

and common in the West Indies. In South America it appears to be common in Colombia, Venezuela, the Guianas and Brazil, but whether it is rare or simply not recognized in other countries is unknown.

It appears to be spread all through Africa, but is certainly rare in West Africa, more common in Central and East Africa, and decidedly more common in North and in South Africa, where there is the celebrated Robbin Island Leper Asylum. There are people who believe Egypt to be the original home of the disease, from whence it spread to Asia and Europe.

**Ætiology.**—The disease is caused by Hansen's bacillus which morphologically has the greatest resemblance to the tubercular bacillus, and is stained by the same methods.

With regard to the cultivation of the bacillus there are three views—

- 1 That it has never been cultivated
- 2 That it can be cultivated as a streptothrix or nocardia
- 3 That it can be cultivated as a bacillus

1 *That it has never been cultivated*—This is still the most generally accepted view.

2 *That it can be cultivated as a Streptothrix*—This is the view held by Bordoni-Uffreduzzi, Babès, Rost, Kedrowsky, Shiga, Hewlett, Rayon, Johnston, and others. The

The growth of the germ is slow and the colonies coalesce into a whitish mass. The inoculation of cultures into monkeys, rats and guinea pigs gives rise, according to Rayon, to leprosy like lesions, with very little tissue proliferation, no caseation, necrosis, no vascular sclerosis, and with presence of numerous acid fast bacilli.

patients by tuberculin.

In 1913 Fraser and Fletcher were unable to confirm Kedrowsky

**LEPROUS LESIONS.** Duval gives importance to a non chromogenic always acid-fast bacillus he has isolated, which grows very slowly,

and only on special media. He states that in addition to this

over the body wherever diseased tissue is found, leave it by the nasal secretion, the tears, the salivary secretion, the sputum, the milk, the semen, urethral and vaginal secretions and by the faces, and are cast off with the scales of skin or the discharge of disin-

tegr.

to b

the bacilli are reported to have been found in *Culex pungens* and *Clinocoris lectularius* by Goodhue of the Molokai Leper Settlement. Finally, notwithstanding one or two observations, the bacillus has never been found in earth, dust, air, water, or food.

and some experiments were thought at the time to be successful. Nicolle produced a hard indolent swelling with a few lepra bacilli by injection of leprous tissue in a *Macacus* monkey. Marchoux and Bourret have made inoculation experiments in a chimpanzee with partial success. Stanziale has inoculated leptotic material in the cornea of rabbits, inducing certain lesions which he has been able to transmit, to a certain point from animal to animal. In rats a peculiar skin disease, somewhat resembling leprosy occurs spontaneously, as observed by Stephansky, Dean, and Rabinowitsch. This has been investigated by Marchoux and Sorel, who have come to the conclusion that it is generally transmitted by contact, and not by parasitic agencies. They have not succeeded in cultivating the bacillus, while Bayon has cultivated a streptothrix very similar to the Kedrowsky strain isolated from human lesions.

With regard to the experimental inoculation of human beings the only case cited as successful is Arning's inoculation of a Sandwich Island criminal in the arm with a leprous tubercle. This man developed a neuritis of the ulnar and median nerves four weeks after the inoculation, a tubercle five months later, the full signs of leprosy two and a half years later, and died a leper six years after the inoculation. It is, however, to be noted that he lived in a leprous country, and that there was leprosy in his family—facts which decrease the importance of the experiment.

There can, however, be no real doubt that the disease is in some way spread from human being to human being. In support of this there are many well known facts—e.g. the case reported by Benson where an Irishman, having acquired leprosy in the West Indies, returned to Ireland and died from the effects of the disease in

about eleven months. During this period his brother not merely lived with him but slept in the same bed and after his death wore his clothes. In about four to five years this brother showed all the typical signs of tubercular leprosy though he had only once been out of Ireland and then only to visit England. Another similar case may be quoted of a person who acquiring leprosy in Tonkin returned to Strasburg and lived with a nephew who subsequently developed the disease. Turning to the evidence of history there is the spread of leprosy throughout Europe and later the rapid spread of the disease in the Sandwich Islands where though existing probably for many years it increased from 1859 when it was hardly known till in 1881 no less than 800 lepers were isolated and it is said that no less than one tenth of the population were affected. Another instance is the case of New Caledonia in which the disease though now common is believed to have been introduced for the first time in 1860 and Pine Island which is said to have been infected from New Caledonia or Mauritius which was infected by a single leper and from which later the island of Rodriguez was infected also by a single leper.

As to individual cases of infection by residence among lepers the most noted is that of Father Damien de Venster who went from Belgium as a missionary to the Molokai Leper Asylum of the

and to day in Norway Sweden and Iceland is in favour of the view that the disease passes from man to man. But it is not wise to hastily conclude that this transference is direct for any of the above cases are easily explicable by the disease being conveyed by food or biting animals. The success of partial isolation might be simply to diminish the chance of infection by these means. Moreover the fact that the attendants of the Hendela Leper Asylum of Ceylon have so far not been known to contract the

Further though  
h suffer from the  
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ty being a source

of infection for never has a child been born in a leprosy condition though it is said that 10 per cent of the children of leprosy mothers become sooner or later lepers themselves.

If the germs are not carried from one person to another by contact sexual or germinal transmission they might still be conveyed by air dust water or food and indeed all these theories have their supporters.

It does not appear likely that it is conveyed by air otherwise it would surely be spread more commonly from patients to attendants in leper asylums. With regard to dust it is quite true that though some persons report the presence of a very few bacilli in earth

taken from places frequented by lepers the majority have failed to find them and again what has been remarked with regard to air also applies to dust. The germs have never been found in the water of the most highly infected places.

Many articles of food have been suspected especially fish and more particularly salted fish—a view which Sir Jonathan Hutchinson has strongly advocated but even he admits that it will not explain all cases particularly its presence in people who have no chance of eating cured fish.

After excluding all these there is still the possibility of the infection being carried by some blood sucking insect. This subject has been most ably discussed by Nuttall who points out that Linnaeus and Rolander considered *Chlorops (Musca) lepræ* to be the active agent while Corredor suspected flies in general Sabrazes insects Joly *Sarcoptes scabiei* and *Pediculi* and Sommer mosquitoes. Nuttall himself says that the possibility of such transmission cannot be denied. Goodhue has demonstrated the bacilli in *Culex pungens* and in *Climocoris lectularius* and Marchoux and Bourret have suggested that some Simuliidæ might be the carriers of the disease. Flies lice bugs fleas ticks etc. have all been studied recently without any great success.

It might be thought that direct inoculation having failed the infection by means of insects would be unlikely. But that is not so because it is well known that the passage of bacilli through another animal may markedly modify the virulence of the germ. On the other hand a great many facts are in favour of the insect spread of the disease—*eg* the infection in a family. The cases cited above as examples of contagion would be easily explicable by the action of an insect as would the effect of isolation in preventing the disease. Moreover the predisposing causes of dirt poverty etc. are also explicable on the same reasoning especially the curious disappearance of the disease in the families of Norwegian peasants emigrating to America where they became much cleaner in their habits. The difficulty of cultivating the germ on ordinary media is very suggestive of its being accustomed to live solely in animal tissues while the abundance of the bacilli in the skin is also suggestive of that being the natural method of leaving the body. Everything in the history of the disease appears to us to favour its spread by animal agency.

Cases of infection by vaccination and varnization are on record. Natives of Ceylon generally state that the disease begins after a bite by a rat.

**Pathology**—According to different theories the bacillus enters the body via the skin the nasal or respiratory mucosæ the alimentary canal or the generative organs.

The list is so comprehensive that it will be obvious that the real method of entry is entirely unknown. On arrival inside the body the bacillus is supposed to come to rest inside a lymph space some where and there to grow and form colonies from whence it can be

seminated through the body, perhaps by the blood and the lymph streams. It must be remembered however, that the

pathogenesis of the lesions is not very well known and there are many

large mononuclear leucocytes

of the type



bacilli surrounded by mucus, forming the 'globi' Beneath the lesion the

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c  
cells at first around the vasa nervorum and later in the perineurium and endoneurium

injury

**Morbid Anatomy.**—The skin lesions which may be found are the tubercles, which may or may not be pigmented areas situated usually in the center of the limb. Tubercles may lie in the subcutaneous tissue in which case it does not form a tubercle. It is yellowish white in colour firm in consistency, and if squeezed usually a little clear fluid can be obtained. It will be noted that the sweat and sebaceous glands and the hair follicles are compressed and as a rule atrophied while vesicles and pustules may occur on the surface, which may be ulcerated and covered with crusts. The macules consist of round celled infiltration with but few large cells which are generally free from bacilli. In the spots which during life were anæsthetic which are derived from the macules the corium is largely converted into fibrous connective tissue which has caused glands and hairs to atrophy and disappear.

The typical lepromatous infiltration may occur, not merely in the skin, but in the mucosæ of the tongue pharynx, larynx, epiglottis, and in the mucosæ of the nostrils.

they are in the septa and a similar infiltration may be seen at times around the bronchi

The ovaries and testes may show infiltrations and fibrosis of the interstitial tissue which destroys the secretory elements and

palmar branch of the ulnar the ulnar the median the peroneal

nerve fibres

In the spinal cord there may be posterior sclerosis and meningitis though it is doubtful whether these are really due to the disease or to some complication. The cells of the posterior cornu have been said to be atrophied as well as those of the anterior cornu in which Lie has found bacilli

In the circulatory organs periarteritis and endarteritis are met with while osteomyelitis necrosis caries and absorption of the bones may be seen and will be mentioned again later. Trophic changes in the joints and perforating ulcers are met with in the nerve form of the disease

acute and at times caseating parenchymatous inflammation or chronic diffuse interstitial inflammation. These are distinguished from similar tubercular affections by inoculation into guinea pigs with negative results

**Symptomatology**—The *incubation period* is entirely unknown and must necessarily remain so until the method of infection and the date of the onset of the disease is discovered hence the statements made by the different observers that it may last for a few weeks or months up to many years. The *method of invasion* is also quite unknown. Sticker suggests that it begins with nasal symptoms—blocking of the nose epistaxis and frontal headache, other observers with skin eruptions. The truth appears to be that so far the initial lesions and their symptoms if any have escaped notice.

Before the eruption appears there are in many cases attacks of

fever of an intermittent or irregular character with a marked feeling of general illness associated with headache and pains in different parts of the body peculiar sensations of cold formication or numbness in various places and above all of abnormal local

### *Bacillus lepræ*

After the general dissemination through the body the bacilli appear to settle mainly in the skin or in the nerves though of course there are many cases in which they settle in both. It is therefore convenient to distinguish the two varieties of the disease first differentiated by Daniellssen and Bocck—viz. lepra tuberculosa or nodular leprosy and lepra maculoanesthetica or smooth leprosy—remembering that the division is artificial and that numerous cases exist which show both forms.

### TUBERCULAR LEPROSY

After repeated attacks of fever the patient has a more severe one during which an erythematous diffuse or macular eruption appears on the face and limbs. The fever subsides and the macule may disappear or thicken and become tubercles which are dermal lesions projecting from the skin or mucosæ in addition to which there are subdermal infiltrations which can be more easily felt than seen. If they disappear it is only for a short time and with a new attack and sooner or later they reappear in the same place and the nodules or tubercles reappear. Each outbreak of nodules is in some cases preceded by an attack of fever with or without an erysipelatous like eruption in the area to be affected associated with enlargement of the lymphatic glands. In our experience however the fever may be absent in many cases.

The nodules may form all over the skin but are most common on the face and limbs. In the former situation they appear on the forehead cheeks along the bridge of the nose lobules of the ears lips and chin and as they increase in size totally alter the appearance of the

disease for Borthen as the result of his investigations concludes that only 8.08 per cent of women and 1.67 per cent of men suffering from tubercular leprosy escape without some form of disease of the nose or the radnexa. Women are however less affected than

men but age shows no influence on the production of eye affections. In tubercular leprosy the eye is attacked by genuine leptotic lesions and secondary infections are rare.

The supraciliary region as has already been mentioned is early attacked and complete madarosis is not uncommon and later, paralysis of the frontalis muscle sets in. The eyelids are often attacked by diffuse or nodular lepromata which may be merely



FIG 72 —LEPROSY SHOWING THE ERYSIPELATOUS-LIKE ERUPTION ON THE ARMS AND FACE

extensions from the disease already in the supraciliary region or may be quite distinct lesions. As a result of ulceration of these nodules the eyelids may be destroyed.

The conjunctiva may be infiltrated leading to hyperæmia or more rarely anæmia and producing lagophthalmos, ectropion and if cicatrization takes place xerophthalmia.

The episclera is apt to become infiltrated along the external

aspect of the corneo-sclerotic junction resulting in white grey or yellow whitish masses which spread round the cornea dorsally and ventrally, and are prone to invade its tissue in the form of a diffuse infiltration which spreads from the outer side towards the pupil.

More rarely small spots form on the corner giving rise to the "keratitis punctata leprosa" of author.

The disease may also spread to the uveal region in the form of an infiltration which causes an anterior or posterior iritis or more



FIG. 222.—TYPICAL TUBERCULAR LEPROSY SHOWS THE LEONINE EXPRESSION, THE THICKENED SUPERCILIARY LIDDS, AND THE MADAROSIS.

rarely, nodules may form in the ciliary body or near the canal of Fontana giving rise to an irido-cyclitis or irido-choroiditis.

He has studied the pathology of these lesions and has shown that it is rare for the optic nerve, the retina, the lens, and the vitreous humor to be affected.

The mucosa of the nose may be attacked with first blocking of the passage, and then when the leproma extends down to the cartilage and ulcerates, falling in or destruction of the nose with much disfigurement of the countenance resulting from the cicatrization which follows the ulceration. The tongue may also be

affected and show numerous tubercles separated by furrows or it may be simply infiltrated. The walls of the mouth and pharynx may become lepromatous which causes mastication and deglutition to be rendered difficult while the same condition in the larynx makes the voice raucous and may impede respiration especially if there is ulceration and cicatrization.

The skin of the hands, arms and legs also shows numerous raised



FIG. 729.—DIFFUSE INFILTRATION OF BOTH CORNEAS AND DEFORMITIES IN THE FINGERS OF THE RIGHT HAND.

tubercles which may ulcerate. The nipple is often infiltrated. The submaxillary, cervical and femoral glands may be enlarged and may suppurate. The testes often become fibrous and

leucocytes is generally normal or according to Bourret diminished while this observer records an eosinophilia in all stages of the disease which he says may at times be quite considerable. There may be leucocytosis during the febrile attack. Neutrophile myelocytes may also be observed. According to our researches the leucocytic formula is extremely variable and is of no help in the



FIG 730—LEPROMA OF THE TONGUE

diagnosis of the malady. In an early case the differential count of 1 000 leucocytes showed polymorphonuclears 52 per cent large mononuclears 38 per cent small lymphocytes 3.3 per cent eosinophiles 6 per cent basophile cells 0.7 per cent. This agrees with Sadi de Buen's observations who also finds that Arneth's index is generally shifted to the left but who also finds that there is no

most marked feature is a great increase in the ethereal sulphates Brinton of Rio de Janeiro has isolated two ptomaines from the urine one allied to choline and the other to muscarine

The nerves may become attacked and the signs and symptoms of nerve leprosy be added to those of the tubercular forming a variety of mixed leprosy

The ulcerations generally become marked towards the end of the disease If treated they cicatrize and produce deformities if left to themselves they suppurate and produce amyloidosis, or becoming phagedenic cause gangrene of the fingers or toes and septic poisoning

Complications in the form of phthisis and amyloidosis appear causing fever cough and expectoration diarrhoea and enlargement of liver and spleen

Unfortunately in the midst of disease of almost every organ of the body the mind is quite clear but the patients are most irritable and it is not surprising that under these circumstances the patients of a leper asylum require considerable tact in management and are often peevish and discontented and that small rebellions occur

### MACULO-ANÆSTHETIC LEPROSY

In this form of leprosy the infiltration takes place principally into the nerves with the result that first the fibres are irritated

formication along with vasomotor disturbance—e.g. flushings of the face glossy skin—and motor disturbance such as twitching

as flat red spots

with other spots forming large areas.



The skin in the affected area becomes anæsthetic the hairs fall out and wrinkles and scales appear. After a time the areas cease to spread the raised margin disappears and the disease becomes quiescent.

Meanwhile the infiltration into the nerve trunk has proceeded to such an extent that a swelling can be easily felt in certain regions—as for example in the ulnar behind the internal condyle in the great auricular over the sterno-mastoid in the peroneal just below the head of the fibula and in other nerves in suitable places if affected. With the destruction of the nerve fibres the hyperæsthetic stage ceases and the anæsthetic stage of the disease begins.

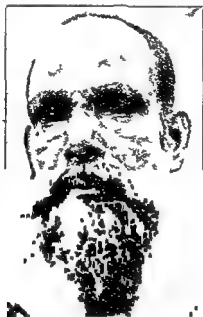


FIG 731 —MACULO ANÆSTHETIC LEPROSY CIRCINATE TYPE

This is to be noticed along the ulnar side of the hand forearm and

the paralysis to the muscles of according to Jeanselme to the sis of the muscles of the forearm the occurrence of dropped wrist is not uncommon

In the lower limb the plantar muscles of the toes may be affected while the spread of the paralysis to the peronei and extensors may



FIG. 732.—MACULO ANÆSTHETIC LEPROSY

Trophic lesions may also appear in the form of whitlows in the fingers and necrosis of the phalanges or instead of this a simple absorption of the bone of the phalanx or metacarpal so that the nail may ultimately appear to spring from the metacarpal (the wrist or even it has been said from the elbow). Similar trophic lesions may appear in the foot. Bullæ may appear on the hands

or feet and when broken may form ulcers. Injuries to anæsthetic areas may also result in ulcers which may be of the perforating type. Fissures may form in the digits hands or feet and more rarely dry gangrene may cause loss of the fingers or toes or greater portions of the limbs and still more rarely the bones of the fingers will soften and become osteomalacic. Trophic lesions of the elbow or knee like Charcot's joints have been recorded.<sup>4</sup>

Jeanselme Bourret and one of us have studied the cerebrospinal fluid and have found in a few cases a lymphocytosis but more usually no cells or bacilli.

The eye is far less commonly affected than in the tubercular variety. Borthen's figures showing that in anæsthetic leprosy no less than 36.83 per cent of the female cases and 26.80 per cent of the male cases escape without eye complications. True leprotic lesions are



FIG. 733.—LEPROSY. ULCER OF THE FOOT

much rarer the eye being damaged by secondary infections brought about by the absence of the lachrymal secretion and the lagophthalmos. The forehead and supraciliary regions are often red, dened and œdematous but complete madarosis is rare while paralysis of the frontalis, corrugator supercili and orbicularis  
ere is  
terior  
f the

cornea may result.

Secondary infections may lead to keratitis, onyx, hypopyon, iritis, irido-cyclitis and destruction of the eye.

Sterility is not so frequent in nerve as in tubercular leprosy. The skin may become infected at an early or late stage of nerve leprosy thus forming one of the types of mixed leprosy.

### Mixed Leprosy

This term has been used to comprise those cases of tubercular leprosy which develop nerve symptoms and those of maculo anæsthetic leprosy which develop nodules as well as those general cases in which both nerve and skin lesions advance hand in hand

### Paraleprosis

Zambaco Von Duhring Gluck Lebœuf and others have drawn attention to various phenomena indicating an attenuated infection in regions in which leprosy has long existed. These

tion

easy and may be readily confirmed bacteriologically by excision of a nodule and microscopical examination of a portion for Hansen's bacillus. The diagnosis of the maculo-anæsthetic cases presents

peroneal and other nerves. The search for the larger nerves

fever and the appearance of fresh nodules. Lebœuf acting on March 19 55

Chujo recommends drawing 5 c.c. of blood from the arm diluting it with 200 c.c. of a 3 per cent solution of acetic acid avoiding contact with the air

The microscopical examination would give more than 50 per cent of positive results

Some authors recommend the blistering of the skin, and examination of the liquid of the blebs for the presence of Hansen's bacillus Thompson recommends for diagnostic purposes the injection of pilocarpine, with the view of discovering dry areas in the sweating skin.

**Differential Diagnosis.**—In countries where leprosy is endemic, other diseases are liable to be mistaken for it One of the diseases most frequently mistaken, as has been pointed out by Powell and others, is frambœsia, which may be recognized by the presence of the *Treponema pertenue* and the frambœsiform appearance of the nodules Leucodermic patches and morphœa are also occasionally mistaken for leprosy, but in such conditions there is no anæsthesia

**Prognosis.**—The prognosis is not good The probability of a permanent cure is slight, but the disease may last a long time Four to twelve years is laid down for the mixed or tubercular leprosy, and longer for the maculo-anæsthetic, and during that time a great deal can be done by appropriate treatment, and indeed, the disease may be stopped for the time being, only, usually, to recur again It has been shown by Lie that, even when all the skin eruptions have disappeared, and the patient is only troubled by anæsthesia, and the atrophy of the muscles and may be thought to be cured, still the bacilli are present in the nerves and spinal cord

Lebœuf from his recent investigations in New Caledonia has come to the conclusion that in a certain number of cases showing slight symptoms, an actual cure takes place

**Treatment.**—So far no specific treatment has been found, though Carrasquilla attempted to prepare a serum by the injection of the blood of lepers into equines, and Abrahams and Hermann by inoculating the juice from lepromata into an animal These sera have been found useless Rost prepared a substance, which he called 'leprolin,' on the lines of tuberculin but, unfortunately, the bacillus he was using was not the leprosy organism Clegg's vaccine and Bayon's extract of Kedrowsky's strain have not been very successful Tuberculin has been tried without success, and,

nc

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*Gynocardia* (*vide infra*), but may be adulterated with other oils, especially that of *Hydnocarpus wightiana* or may be altered by being expressed when heated It should be rubbed into the diseased patches, and also given internally in doses of 5 to 10 minims, working up gradually to 30 to 60 minims, in capsules, or in a pill with tragacanth and soap, or in an emulsion, or as Engel antileprol capsules To remove the after-taste a lime can be sucked This treatment must be persisted in for a very long time, and should be

combined with hot baths and in nerve cases with doses of strychnine  $\frac{1}{10}$  grain three times a day which may gradually be increased. After a length of time it is as well to make a slight break in the treatment to prevent toleration and therefore Unna's ointment of ichthyol 5 per cent, salicylic acid 2 per cent, and pyrogallol

camphorated oil and Mercado has combined the mixture with the resorcin formula of Unna. Heiser's present formula is—

Chaulmoogra oil	60 c c
Camphorated oil	60
Resorcin	grms 4

Mix and dissolve with the aid of heat on a water bath and then filter.

The injections are made in the gluteal region at weekly intervals in ascending doses one to five or ten cubic centimetres. During the treatment the patient takes a hot sodium bicarbonate bath (2 per cent) every other day. The results are fairly satisfactory. *Sodium Gynocardate*—Rogers recommends the intravenous in-

pills after meals or by subcutaneous injection.

Neumann has advised the combination of salve and theonin with Chaulmoogra given either by the mouth or hypodermically but the advantages are doubtful and Hollmann has recommended the use of eucalyptus oil in conjunction with opia leaves (*Jambos malaccensis*) or with Chaulmoogra oil.

*Squize of Chaulmoogra Oil* — A dose of 10 to 20 c c

*Cod liver Oil and Sodium Morrhuate*—Cod liver oil is occasionally beneficial. Rogers recommends an intramuscular injection (1 to 3 c c) every other day of a 3 per cent solution of sodium morrhuate.

Other remedies are legion—e.g. X rays have been well spoken of but must be pushed to the extent of almost burning the patient. A 10 inch spark-coil with a bifocal tube situate 7 to 10 inches from the lesion has been used. Hypodermic injections of perchloride of mercury, as advocated by Crocker (0.01 gramme every other day) have been found satisfactory at times—a treatment which we recommend in cases at the very beginning of the disease.

Cashew nuts (Beaupertney treatment) have been applied to the lepromata with the idea of local caustic action. Thyroid gland, salol, salicylates, arsenic, Turjun oil, chlorate of potash, sodium hypodermic injections of iodolorm have all been tried and found wanting.

Hypodermic injections of nastin have been tried. This is a fatty principle extracted by Deycke from cultures of a streptothrix (*Streptothrix leproides*) which he found in the nodules of leprotic patients. The nastin is combined with benzoyl chloride and made into ampoules with sterilized olive oil by Kofke and Company of Biebrich on the Rhine. Each ampoule contains from 0.0005 to 0.0002 gramme of nastin which is to be injected once a week and in the small doses produces no local reaction, but in the larger dose causes considerable local inflammation. Deycke's views as to the method of action are that the nastin attaches itself to the lepra bacillus and then the benzoyl acts on the bacillus damaging it by removal of its fat when the normal fluids of the body complete its destruction.

Wise and Minett and others have reported unfavourably on this treatment but recommend benzoyl chloride in petroleum oil as a valuable nasal spray or punct as it renders the discharge from the nose free from bacilli.

Castellani and Woolley and more recently Nicholls have tried a vaccine prepared by triturating nodules rich in bacilli in salt solution or broth then filtering through gauze and finally heating to 60° C for an hour.

Suga recommends intravenous injections of a solution of potassium cuprocyanide.

Surgical treatment on the ordinary lines is required for ulcers, whitlows, etc. Eye lesions should be treated as though the disease was non-leprous and should not be neglected. Grossmann thinks that leprous infiltration might be arrested by the production of

provided with  
 ters should be  
 that they are  
 supplied with some form of light work and amusement and the institution of rural colonies provided they are well supervised is to be recommended.

**Prophylaxis**—Beyond isolation and antiseptic precautions after

The financial burden of heavy. Hence the difficulty of carrying out this very necessary method of protection completely and efficiently.

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## CHAPTER LXX

# HISTOPLASMOSIS

Definition—History—Ætiology—Pathology—Morbid anatomy—Symptomatology—Treatment—References

**Definition** — Histoplasmosis is an acute specific infection caused by *Histoplasma capsulatum* Darling (*Cryptococcus capsulatus* Darling)

**History** — This disease has been described by Darling who also found the parasite but being first discovered post mortem the clinical signs are rather deficient. The first case was in a negro who three months previously had come from Martinique to the Canal zone of Panama.

**Ætiology** — The disease is caused by the parasite *Histoplasma capsulatum* Darling which was at first considered to be a protozoon but is now believed to be a fungus (*Cryptococcus capsulatus* Darling p 1076)

**Pathology** — The parasite infects epithelial and endothelial cells of the lungs liver and spleen. It also exists free in these organs. In the lungs it gives rise to pseudo tubercles resembling miliary tubercles.

CHRONIC THE PARIELES DIAPHRAGM BONE MARROW AND BLADDER WALL

found to consist of collapsed and were

**Symptomatology.**—The symptoms closely resemble those of Indian kala azar there being irregular fever with enlargement of the spleen and liver and severe anemia with marked leucopenia

**Treatment.**—Nothing is known as to the treatment or prophylaxis.

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## CHAPTER LXXI

# BERI-BERI AND EPIDEMIC DROPSY

Beri beri—Infantile beri beri—Epidemic dropsy—Potter's disease—References

### BERI-BERI.

**Synonyms.**—Polyneuritis Endemica Neuritis Multiplex Endemica Hydrops Asthmaticus Synclonus Beriberi Myelopathia Tropica Scorbutica Para

**Etymology.**—The word *beri-beri* is said to be derived from the Sinhalese term meaning 'cannot' which is used as a phrase which means I cannot employed in the sense that the person is too ill to do anything. There is another Sinhalese word which may equally be translated as 'cannot' but this means that the person is unwilling to do something not that he is too ill to do it. It is possible that the above interpretation of the word is correct, for Ceylon has long been in the hands of Europeans—e.g. Portuguese, Dutch, and English—and therefore a term used therein would be widespread. There is no doubt that the word covered a large number of diseases from which a definite pathological entity has gradually been separated out. It is to be noted however that—at all events at the present time—the disease does not exist endemically in Ceylon where there are only imported cases.

**Definition.**—Beri beri is an acute or chronic endemic or epidemic disease of unknown causation which is characterized by degeneration of many peripheral nerves especially the vagi, the phrenics and those of the limbs, associated with gastro-intestinal disturbance,

to the second century of the present era and is minutely described

In 1758-59 Bontius was the first European doctor to give an account of the disease which he described under the term '*beri-beri*'

Later Tulpius a Dutch physician also described the symptoms of the disease as seen in a person who had returned to Holland from the Indies. From that time the literature on beri beri has grown until it has reached enormous proportions but unfortunately there is no doubt that until recently several diseases especially ankylo-

stomiasis

In 1847 Carter gave a description of the disease indicating asthma as the principal feature probably because of the cardiac dyspnoea. In 1873 Fayrer laid great stress on oedema as the important clinical feature. In 1877 Wernich defined the disease kakke in Japan while van Meedervoort showed that it was the same disease as beri beri. In 1886 Kynsey published an account of the anæmia or beri beri of Ceylon by which he really meant ankylostomiasis hence the idea that beri beri was due to *Ancylostoma duodenale* and hence the large number of cases of so-called beri beri in Ceylon years ago.

The scientific and clinical study of the disease has been largely due to the work of Oudenhoven, Da Sylva Lima, Overbeck de

treated in the section on the causation.

Beri beri is in certain regions a most serious disease. Thus Braddon says that in the Straits Settlements and Malaya where the population is only 1,250,000 no less than 150,000 cases of the disease with 30,000 deaths have occurred in Government hospitals and infirmaries in the last twenty years but he says that only

continuously disabled by it. In 1904-05 it is said that 24 per cent of the entire sick and wounded in the Japanese armies—about 85,000 men—were also disabled by it. It is obvious

cost of the disease to the Government of Malaya as £10,000 per

the dietary (*vide* Chapter IV p. 109)

In 1913 Cooper and in 1917 Chuck and Hume studied the subject of vitamins the two latter especially dealing with beri beri vitamin

**Climatology**—The endemic centre of beri beri appears to be Eastern Asia Japan China the Philippine Islands Indo-China Java and to Plehn Senegal have not exist Isolated epidemics sporadic or imported cases occur in England Ireland and other parts of Europe the United States Canada and in Ceylon In the last named place it is now almost entirely absent and indeed it is possible that many of the cases reported years ago may have been ankylostomiasis or some other disease Imported cases from India and China are frequently met with in Colombo owing to its possessing a large harbour where

nyu and believed at one time to be beri beri has been demonstrated to be Malta fever In the tropics it is said to occur more often in the cool wet months

**Ætiology**—The causation of beri beri is at the present time believed to be due to the deficiency of some essential substance in the food but as may be imagined in such a widespread and fatal disorder the investigators have been many and hence the theories which

**A Physical Cause**—The older writers like Davy considered that it was brought about by some unusual state of the atmosphere but they said honestly that this was merely a cloak for the ignorance of the true cause

**B Chemical Causes**—The chemical causes which have been advanced are —

- 1 Arsenical poisoning (Ross)
- 2 Oxalate poisoning (Trentle n)
- 3 Carbon dioxide poisoning (Ashmead)
- 4 Food poisoning —
  - (a) Ichthyotoxism (Grimm and Mura)
  - (b) S totoxism (Eijkman Vorderman Yamagata Van Dieren) rice (Gelpke Braddon) lathyrism (Le Roy de Méricourt)
- 5 Some deficiency in the food —
  - (a) Deficient nitrogenous complex (vitamine) (Eijkman Fraser and Stanton)
  - (b) Deficient nitrogen (Takaki)
  - (c) Deficient fat (Brémond and Laurent)
  - (d) Deficient vegetables together with an infection (Fales)

produced in fowls by Eijkman by feeding them with cooked rice and it is possible that neither oxalic acid nor rice but an infection was the cause of the disease which may of course be quite different from true beri beri.

3 CARBON DIOXIDE POISONING—Ashmead believes that the disease is

of the same nature as that which is shown that is the case at Kuala

eaten rice

Travers details an interesting observation on this point concerning an

5 SOME DEFICIENCY IN THE FOOD—(a) *Deficiency of Certain Nitrogenous Complexes*—There is a growing tendency to consider that certain diseases—e.g. beri beri polyneuritis of birds epidemic dropsy scurvy experimental scurvy infantile scurvy and ship beri beri—are diseases due to the deficiency in some essential substance in the food. These diseases Funk classifies together as deficiency disease and characterizes them by certain genera

prev on hea tee  
 11 f u o u j u j s  
 The difference between these two forms of rice is that the white rice is deprived of its subpericarpal layers (vide pp 104 and 105) by the process of milling

These layers contain a substance called by Funk beri beri vitamin which is probably a base belonging to the pyrimidine group and has the formul—



tissue and if it is not present in the dietary must be supplied by the



animal body and if this fails the nervous tissues begin to break down and as a result the signs and symptoms of beri beri appear

Fraser and Stanton believe that the phosphorus content of the rice is a good index as to whether it is harmless or harmful. A safe rice yields more than 0.4 per cent of phosphorus pentoxide while a dangerous rice yields less than this figure. Chamberlain and Vedder have suggested that potassium should be used instead of phosphorus for standardization purposes.

With regard to these findings there is an almost unanimous support from

believing that  
forward the claim  
their Native States

decrease in the general incidence of the disease in the islands and four months before the use of under-milled rice was introduced into the dietary. They consider that the reduction was either due to unknown causes acting coincidentally with a reduction in the amount of rice in the dietary together with the addition of a legume or was due directly to these dietetic changes.

Eijkman, Braddon, Fraser, Stanton, Vedder and Chamberlain have done sufficient work to make imperative the use of brown rice cooked in ordinary vessels and the exclusion of the white rice as a staple article of food.

Edie, Evans, Moore, Simpson and Webster have separated an antineuritic base called torulin— $N(CH_3)_3C_4H_7O_2(HNO_3)$ —from yeast and Thomson and Simpson have noted rapid recovery of patients placed on a full diet and given 1 ounce of yeast and 200 grammes of katjangdo-beans daily.

Heiser reports that after being present for five years in the Culion Leper Colony in the Philippines beri beri disappeared in nine months on a dietary of unpolished rice.

'anti beri beri' as the first term covers the polyneuritis in fowls. Neither vitamin has yet been isolated in a pure condition. Pigeons deprived of anti beri beri vitamin develop acute polyneuritis in fifteen to twenty-five days. The principal source of this vitamin is the seeds of cereals and pulses. In the former it is mainly de-

Antineuritic vitamins cannot be expected to survive in tinned or sterilized foods hence the necessity in armies to supply vitamins from other sources.

The following objections have been raised against the food theory —

- (1) Beri beri may occur in people who do not feed on rice. We have seen an epidemic among officers on a man-of-war.
- (2) —
- (3)
- (4)

Fraga, having failed to produce beri beri in prisoners fed on polished rice

196 grammes fats 43 grammes, and carbohydrates 775 grammes, and on this dietary the warship *Takakura* was sent the same cruise taking 287 days and only suffered from sixteen cases of the disease. Takaki attributed the success by corn or creased fruit alone may dietary was applied to the Japanese Army and Navy and beri beri decreased enormously. But Baelz has pointed out that this decrease was associated

according to Voigt's diet, to have been at least 94 grammes fats 45 grammes, and carbohydrates 400 grammes; or, according to Moleschott's diet, nitrogen 256 grammes; carbon, 3 789 grammes; hydrogen, 143 grammes, sulphur, 23 grammes, salts 172 grammes—*L. N. C.* 1 0-15

The epidemic of beri beri now began December 1901 52 cases and 2 deaths January 1902 169 and 12 February 1 087 and 16 March 5 6 and 15 April 327 and 15 May 310 and 19 June 451 and 17 July 233 and 33 August 571 and 24 September 522 and 31

On October 20 the diet was again changed and this time proteids were 101 71 grammes fats 19 37 grammes carbohydrates 395 73 grammes salts 79 13 grammes including 119 07 grammes of potatoes and 255 15 grammes of rice Nitrogen was 209 8 grammes carbon 3 816 2 gramme Hydrogen 70 4 grammes sulphur 17 2 grammes and salts 185 grammes—  
N C I 13 4

In October there were 579 cases and 34 deaths November 476 and 8

in common in the two regiments—viz both brought the disease with them—but was unable to find any source of infection parasitic or otherwise

(e) *Deficient Phosphorus*—Schaumann and others have maintained that deficient organically combined phosphorus in the uncured rice is the etiological factor

(f) *Deficient Cholesterolin*—Christensen has treated cases with injections of cerebrin with good results and with 5 per cent cholesterolin in olive oil with better results

6 AN INTOXICATION FROM A GERM LIVING OUTSIDE THE BODY—Manson brought forward the hypothesis that a germ may live in the soil the house or the ship occupied by the human being under certain conditions of tem

necessity

Manson rightly points out that these children must have been poisoned

But of course

ups in which it  
e is also evidence

intoxication may produce peripheral neuritis but the epidemics of the

American negroes Americans and Europeans but the disease fell most

Further he points out that infection by air and water can be excluded as in

Island to be useless As regards parasites Durham and Daniels are both  
 agt nat mosquitoes as being the cause—and indeed this is hardly likely—and  
 also agt nat bugs

ANIMAL PARASITES —

(a) Protozoa

- 1 Plasmodium in the blood (Glogner)
- 2 Protozoon in the urine (Hewlett and Korté)
- 3 Hæmatozoa in the blood (Fajardo and Voorthu)

(b) Vertebratelmintes

- 1 Some form of *Trichinella* (Gelpke)
- 2 *Trichuris trichiura* (Ertz and Lynsey)
- 3 *Ancylostoma d.odenata* (Fruenl and Kynsey)

VEGETAL PARASITES—FUNGI—

(a) Coccales

- 1 Cocci in the alimentary canal etc (Dangerfield)
- 2 Diplococcus in the urine (Tazuka)
- 3 Diplococcus from the blood and urine or organs post mortem  
 (Okata and Kokubo)
- 4 Four kinds of cocci (Nasso and Morelli)

(b) *Coccus and Bacillus*

Pleomorphic organism obtained by Pekelharing and Winckler  
from the blood

Bacilli and cocci by Lacerda

(c) *Bacilli*

1 Bacillus by Taylor

2 Bacillus by Rost

3 Bacillus by Ogata

4 Three kinds of bacilli by Nepveu

5 Bacillus by Eccke

Toxins from a bacillus in the alimentary canal (Hamilton Wright)

(d) *Fungi higher than Bacteria*

Mouldy rice (Hose)

*Protozoa* — Glogner's parasites were similar to malarial parasites but were  
distinguished therefrom by being found only in splenic blood by being

nor do they correspond to Glogner's

*Nemathelminthes* — Gelpke suggested that the disease might be due to a  
*Trichinella* in fish but he has withdrawn this. *Trichinuris* and *Ancylostoma*  
need not seriously be considered as this idea arose from a misunderstanding

*Fungi* — Pekelharing and Winckler's bacillus has also been found by Hunter

Chalmers and Archibald separately have found fungi in the organs

but the others await discovery

and at  
to one  
in the

According to Gales the Filipinos suffered most severely the Chinese were almost exempt only one or two contracting the disease while the Americans were entirely free

With regard to age it is most commonly met with in young adults between fifteen and thirty years but it has also been noted in babies at the breast and in old men It is more commonly met with in men than in women

Occupation has been carefully investigated by Hunter and Koch in Hong Kong and they conclude that the disease is universally present throughout the community but especially affects the working classes while the professions the merchants and the leisured classes are practically but not entirely exempt Other predisposing causes are disturbance of the soil and a high atmospheric temperature

**Important Features**—The peculiar features of the disease are

due to the vasomotor disturbance

Durham considers that the urine indicates a serious diminution of the metabolism

**Morbid Anatomy**—The morbid anatomy and histopathology have been studied by numerous observers among whom the investigations of Wright Duerck and Scheube must be especially mentioned In acute cases there is always some œdema but at times this may be excessive and the veins of the neck are swollen Hypostasis is always well marked but may be excessive especially about the face There is often froth at the mouth In chronic cases the body is pale and may be swollen with dropsy or emaciated

In the former the post mortem rigidity develops quickly, and is well marked. In the latter, however, it is not so marked. On cutting into the body the subcutaneous tissues are usually oedematous and the veins are filled with dark fluid blood. There is a varying quantity of serous fluid in the abdomen, the chest, and the pericardium, and there may be petechial hæmorrhages under the visceral pleura and pericardium.

The thro  
but they

The mucosa of the trachea and bronchi may be oedematous, with the lumen full of fluid. The lungs may be congested and oedematous and may contain little air.

The right side of the heart is always greatly dilated in acute cases and is also hypertrophied in older cases but it is rare for the left ventricle

moderate ext  
tion, the stria  
of the fibres

be seen and there is a round-celled infiltration beneath both endo-  
id by Scheube  
vagi)

of the heart is  
damaged in acute cases the cells of the bulbar nuclei and the nucleus ambiguus on both sides being swollen with excentrically placed nuclei and a disappearance of Nissl's bodies in the processes, and to a less extent at the periphery. These changes may also be seen in the first and second pair of the thoracic ganglia, and in the intrinsic ganglionic cells of the heart, while the vagal nerve-endings show rounded droplets of altered myelin (neurokeratin<sup>2</sup>), especially near the nodes. In chronic cases only the vagi may show degeneration, the ganglia in the heart being normal.

In acute cases the stomach and duodenum are markedly affected,

pylorus and the duodenum

Microscopically there is an acute congestion with round celled infiltration with according to Herzog, a very large number of eosinophile cells and necrosis of the glandular epithelium. The cells of Auerbach and Meissner's plexuses are degenerate, and the nerve fibres in the stomach and duodenum also show signs of degeneration. In chronic beri beri these gastro-duodenal signs are absent.

In an acute case the lymphatic glands near the stomach and duodenum are enlarged and congested. The liver is generally enlarged and congested, and at times in a nutmeg-like condition and, according to Hewlett and Korté, there may be extensive hæmor-

rhagic patches; Scheube and Plehn draw attention to a round  
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 be  
 als

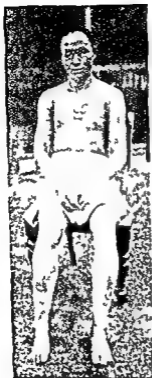


FIG 734 --BERI BERI DROPSICAL  
 OR HYPERTROPHIC FORM



FIG 735 BERI BERI DRY OR  
 ATROPHIC FORM

may be pigmented and congested with vacuolated cells. The kidneys are usually swollen and hyperæmic and there may be

brain may be hyperæmic and there may be some hyperæmia of the brain substance itself and increase of fluid in the ventricles, but



have described degeneration of the cells of the posterior spinal ganglia and anterior cornua of the lumbar cord, together with atrophy of the peripheral nerves. The changes in the peripheral nerves are of the following nature:

may be injected and hæmorrhagic. Scheube and Baelz first showed that these nerves were degenerated, the muscular branches of the nerves of the limbs being most affected, but even the fine sensory cutaneous branches were also attacked. The changes in the autonomic nervous system do not appear to have been closely studied, though several observers have recorded changes in the ganglia, and in the cardiac and other plexuses.

The degeneration of the nerve fibres has been carefully studied by Scheube, Baelz, Hamilton, Wright, and Duerck. The neurofibrils become wider while its sheath becomes vacuolated, and the neurokeratin network condenses into small rosary like masses or larger lumps, somewhat regularly arranged. Later the medullary sheath breaks up into spherical or elongated masses separated by clear intervals. The axone undergoes first chemical changes and then appears like a wavy cord, or as a series of comma like segments or twisted up into a coil. Finally, both axone and medullary sheath disappear, while Schwann's sheath collapses and so the nerve fibres become

nervous system. When fully degenerated the nerve may consist simply of connective tissue. Regeneration has not been observed, but must occur—at all events to some extent—in cases which recover.

The muscles show atrophy and normal fibres are few. The

connective tissue of the muscle increases in amount. The bone-marrow is said to be normal.

unknown and so unknown.

The disease is insidious in its onset and is characterized by gastro-intestinal cardiac and nervous symptoms. For purposes of description three types may be recognized—viz (1) the acute pernicious form, (2) the typical form (3) the rudimentary form.

**1 Acute Pernicious Form**—The acute pernicious form may exhibit itself in several ways. The most acute is when the person without previous illness suddenly dies and the autopsy reveals that he has died of beri beri.

The more usual history is that the patient feels a disinclination for food followed in a short time by a sensation of depression or pain in the epigastrium and nausea. Tenderness is evinced if pressure is made over the pylorus or duodenum while the throat is seen to be congested. The temperature is usually normal though

into the pericardium pleura and peritoneum.

There may early be found patches of anæsthesia or hyperæsthesia particularly in the course of the anterior tibial and musculo-cutaneous nerves. Paralysis now appears and may be slight or may be so extensive as to prevent all voluntary movements and at the same time the anæsthesia may increase considerably.

Sooner or later the cardiac symptoms become worse if the pericardial distension has not been relieved.

**Failure.** During this dying agony the mind is clear almost to the last. The duration of such an attack is from twelve hours to a few days.

■ **The Typical Form**—The typical form of the disease begins

œdema along the lower extremities. There is a sense of heaviness or difficulty in walking which appears or increases in the morning. The gait is peculiar but generally being raised with a jerk.

often with the legs wide apart, in order to give stability to his

mostly affected, and that often there is a tendency for the foot to assume the equino-varus position, and that ankle-drop is often seen

The forearms may also be paralyzed, and wrist-drop, with great

is a partial reaction of degeneration, but if no dorsal movement is capable of being made, then the reaction of degeneration will be fully developed. The paralysis spreads to the muscles of the calf, the muscles of the thigh, and the gluteal region, and to those of the hand and arm, then to the abdomen, the diaphragm, the intercostals, and the larynx, and in rare cases to the intra- and extraocular muscles.

Fletcher has drawn attention to jongkok or squatting test. The person places both hands on the top of the head and slowly squats down on his heels and then rises up again. In beri beri this cannot be performed.

Associated with the paralysis there is at first hyperæsthesia especially in the calf muscles, paræsthesia as already mentioned, and partial anæsthesia which may be characterized by saying that

segmental or nerve areas but are remarkably patchy. The nerves

heard, and reduplication of the second sound is common, particu

LESLIE

The blood does not show much abnormality beyond a certain amount of anæmia. The differential leucocyte count contains about

58 per cent of polymorphonuclears 36 per cent of lymphocytes  
4 per cent of mononuclear leucocytes and 3 per cent of eosino-  
philes

The *cerebro spinal fluid* is generally normal. Lumbar puncture  
rarely shows in our experience any alteration of pressure although

but  
tity  
rides  
and  
albuminuria being rare

*Dropsy* may or may not be present but some *œdema* most  
marked anteriorly in the legs is a practically constant symptom.  
It usually begins along the shin in the form of a rather solid *œdema*.  
It may spread over the legs into the scrotum on to the abdominal  
wall and into the face and arms and is characterized by being at  
times in peculiar localized patches and if it takes place in the  
muscles gives rise to an appearance like pseudo-hypertrophic

authors the two forms being merely the early and late stages of the  
disease

The tongue is usually clean the throat may be slightly con-  
al  
re  
he  
d  
he

voice rendered raucous or lost. The lungs may be *œdematous*.

In this condition the patient may remain at times better at  
times worse for weeks and months and may proceed slowly to  
recovery with of course deformities if paralysis remains or may  
die suddenly of cardiac failure when sitting up or getting out of

be first increase and then diminution of the knee jerks patches of  
anæsthesia some muscular weakness some gastric catarrh and  
general malaise. Repeated attacks may occur.

**Complications**—Whenever fever develops in a beri beri patient  
there is sure to be a complication. The most common are tuber-  
culosis dysentery and malaria

**Sequelæ**—Many authors do not believe in sequelæ to beri beri but certainly there may be the contraction left after the paralysis and anæmia and attacks of palpitation may occur

**Diagnosis**—The principal positive signs on which to base the diagnosis are (1) Loss of knee jerks (2) patches of anæsthesia and occasionally hyperæsthesia on the legs (3) pain on pressing the calf muscles (4) œdema along the shin (5) absence of marked albuminuria and (6) the absence of fever

Suspicious ear  
desire for light  
and duodenum

Severe  
all the di  
by the l  
by the a  
colic and the blue line of the gums

Secondly *dropsies* due to heart disease are recognized by the murmurs and the history of rheumatic fever or other infectious disease kidney disease by an examination of the urine ankylo stomiasis by the ova being found in the fæces epidemic dropsy by the fever and the absence of anæsthesia and paralysis malarial cachexia by the enlargement of the spleen and perhaps the parasites in the blood and kala azar by the enlarged spleen and liver

Thirdly certain *diseases of the spinal cord* myelitis by the loss of control over the bladder and rectum locomotor ataxy by the Argyll Robertson pupil pellagra by the skin eruptions

Fourthly *certain intoxications* such as ergotism by the gangrene and the history and lathyrism by the absence of tender muscles

**Prognosis**—The acute pernicious form is always fatal the rudimentary never The general mortality varies in different countries as follows—

	Per Cent
Sumatra	60 to 70
Hong Kong	48 6
Malaya	19 7
Java	2 to 6
Japan	2 5 to 3 5

An attack does not confer an immunity On the contrary it  
to another attack

**Treatment**—The treatment is essentially symptomatic the

patient being placed in bed, and care being taken to avoid anything which is likely to bring on cardiac failure. Especial care is required for the patient with a severe attack.

If the attack is severe, with great embarrassment of the right heart, it has been advised to perform venesection and remove some eight ounces of blood. Oxygen is useful during the attacks of dyspnoea. For the paralysis strychnine should be administered, and the muscles massaged to prevent atrophy and cramps. Electrical excitation is also good.

If possible, the patient should at once be removed from the place in which he is supposed to have acquired the disease.

Braddon strongly recommends atropine either as the alkaloid in hypodermic injections of  $\frac{1}{100}$  to  $\frac{1}{50}$  gram, according to the urgency of the symptoms, or in the form of the tincture of belladonna. He considers that the atropine is specially useful in cases of dyspnoea due to cardiac failure and pulmonary embarrassment, while he gives a mixture of tincture of belladonna  $\frac{1}{2}$  drachm, tincture of scilla  $\frac{1}{2}$  drachm, and citrate of potash  $\frac{1}{2}$  drachm, in 4 ounces of water three times a day for three or more days.

With regard to after-treatment any deformity such as club-foot, must be rectified, as described in works on orthopedic surgery. Fraser and Stanton have prepared a remedial agent on the lines indicated by their researches and this should be tried when available. Only harmless rice—i.e., brown rice with more than 0.4 per cent of phosphorus pentoxide—should be given to the patients and care should be taken that it is cooked in ordinary pots, and not under pressure. Thomson and Simpson recommend a full diet with 1 ounce of yeast and 200 grammes of katjangido beans daily. Chamberlain and Vedder recommend that 5 c.c. of an extract of rice polishings be given daily to infants suffering from beriberi.

Should be discontinued from this diet. Careful nursing is necessary because of the danger of cardiac failure and good hygiene is also necessary.

**Prophylaxis.**—Rice should be avoided as a staple article of diet, but if it has to be used it should be in the form of the Indian country rice or paddy variously described as the cured stale unpolished, or parboiled rice. Great care should be taken with the cooking, for a good rice can be converted into a harmful rice by cooking, which should always be performed in ordinary pots and never under pressure by steam. With regard to the different kinds of rice, Schuffner and Kuenen find that Rangoon rice contains 0.42 to 0.46 per cent of  $P_2O_5$ , while Sum and Java rice is much lower. They maintain that there should be a rice reform, and that a

minimum legal limit of 0.5 per cent  $P_2O_5$  for dry rice should be imposed or failing this the substitution of other foods to make up the deficient ingredient and a strict control of cured rice—i.e. white rice. Pregnant and nursing women especially should have a liberal diet and harmless rice.

A good nourishing diet is most important.

Good hygienic surroundings—i.e. good ventilation, the avoidance of overcrowding, plenty of sunshine and exercise in the open air—may be mentioned.

It is as well to thoroughly disinfect with Clayton gas or sulphur and formalin any room in which beri beri patients have been living or any infected house or ship.

#### SHIP BERI BERI

*Synonym*—Norwegian Beri beri. Some authorities consider ship or Norwegian beri beri to be a separate entity from tropical beri beri and believe it to be a deficiency disease taking an intermediate position between tropical beri beri and scurvy. Clinically however the condition is identical with tropical beri beri and runs the same course.

#### INFANTILE BERI-BERI

a  
dy

Investigations show degenerations in the vagi phrenics, intercostals and anterior tibial nerves but not so extensive as in adults. Chamberlain, Vedder, Andrews and others conclude that this is an infantile beri beri due to some deficiency in the mother's milk and find that it causes 56 per cent of the infantile mortality in the Philippines.

#### EPIDEMIC DROPSY

*Synonym*—Acute anæmic dropsy.

*Definition*—Epidemic dropsy is an acute infectious disease of unknown cause characterized by fever, dropsy, an erythematous eruption and sometimes cardiac symptoms but without paralysis or anæsthesia.

*History*—In 1876-77 there was a great famine in Southern India during which a dropsical disease at the time called beri beri was noted. It is possible that this dropsical disease was conveyed in some way from Madras to Calcutta for in 1877 there was an outbreak of epidemic dropsy for the first recorded time in that

Calcutta to Shillong, Dacca and South Sylhet and to Mauritius by means of labourers passing through that town. Mauritius

beri beri. Greig has come to the conclusion that it is a deficiency disease.

**Climatology**—The disease is met with in India and Mauritius.

it there  
it is a  
post dysenteric anæmia or hydræmia (3) that it is due to nitrogen starvation (4) that it is due to eating Burma rice the action of poisoning person to

the fact that the disease is epidemic and spreads apparently by the agency of

a rust or fungus on rice.

**Pathology**—No remarks can be made on this part of the subject.

**Morbid Anatomy**—There is subcutaneous œdema and fluid in the peritoneal and pleural cavities. The mouth and pharynx are œdematous and the œsophagus may be ulcerated. The stomach is very congested and may show hæmorrhagic patches and the

time sometimes as long as a month. Associated with the fever and indeed the most

centimetre and a hæmoglobin count of 54 to 65 per cent. The



colour index is said by other observers to be about normal and the proportion of white to red cells to vary from 1 to 430 at the invasion to 1 to 384 in the course to 1 to 615 at the end. Leucocytes are apparently always increased in number but only slightly. The differential count is —

	Per Cent.
Polymorphonuclears	60.2
Lymphocytes	21.4
Mononuclears	11.7
Eosinophil cells	6.7

No animal or vegetal parasites have been discovered in blood which is sterile.

The condition of the urine is described as very variable but there is no albumen and no casts are to be found.

Effusions into the peritoneal and pleural cavities may take place. The alimentary canal is early irritated and vomiting and diarrhoea are common occurrences. A rash appears early on the extremities in the dropsical areas; it is usually erythematous or 'measly' but vesicles and hæmorrhages may be seen. Anæmia as remarked is progressive during the disease and there may be cardiac dilatation with hæmic murmurs. In severe cases there may be cough and dyspnoea due to œdema into the lungs. Recovery is the rule but death may occur from cardiac or pulmonary complications.

**Sequelæ** —The only sequela so far observed is cardiac weakness.

**Diagnosis** —Epidemic dropsy shows the following characteristic signs: dropsy, slight fever, diarrhoea, rash, anæmia, and no albuminuria.

Its diagnosis from *beris beris* is based upon the presence of fever, the persistence in some cases of the knee jerk, the lack of paralysis of painful muscles, and of anæsthesia. Some authorities maintain and no rash, and

in the aged the

ation of calcium chloride or iron and strophanthus are the only remedies usually required. High temperatures should be treated by diaphoretics, quinine, and sponging.

**Prophylaxis** —Segregation and disinfection are recommended but no rational prophylaxis can be advised so long as the cause remains unknown.

### POTTER'S DISEASE

symptoms. Both sexes are attacked generally after puberty and in rural districts. Whole families may be attacked.

## WAR ZONE OEDEMA

In soldiers prisoners of war and refugees having a very scanty and unsuitable diet an oedematous condition of the legs and feet is far from rare. It is a deficiency condition more closely related to scurvy than true beri beri. It should be differentiated by blood examinations from a clinically similar condition due to malaria.

## TROPICAL OEDEMA

The condition is not related in any way to beri beri or scurvy and is not influenced by a change of diet. It disappears rapidly on going to the hills. The same condition has been recently recorded by Marshall from the Red Sea and Bagdad.

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CHAPTER LXXII  
TROPICAL POISONINGS

General Remarks—Ackee poisoning—Onychia—References

GENERAL REMARKS

Other forms of poisoning such as arrow poisons animal poisons trade poisons can be found in Chapters XI (p 180) and XII (p 187)

ACKEE POISONING

Synonym —The vomiting sickness of Jamaica

Definition —An acute and often fatal illness occurring mostly

tioned the ackee without however being definite as to its causal effect

In 1912 Potter after considering cerebro-spinal meningitis, vomitine poisoning, ackee poisoning and cassava poisoning came to the conclusion that it was a phase of yellow fever

In 1913 Scott suggested that it might be fulminating cerebro-spinal meningitis and in the same year Seidelin opposed the yellow fever and the meningitis views and believed it to be a local disease

in a single case. Persons taking soup or pot water made with ackees developed symptoms in two hours and death nearly always resulted

The ackee is the fruit or aril of *Blighia sapida* Koenig belonging to the natural order Sapindaceae and being a native of West Africa is merely an introduced plant in Jamaica

Only unsound ackees cause the symptoms and an ackee is unsound (1) when it is unopened (2) when it is picked from a decayed, bruised or broken branch (3) when it has been forced open (4) when it has a soft spot

In 1917 Scott confirmed this view as to the causal effect of the ackee and by his experiments upon animals demonstrated the nature of the vomiting sickness

**Climatology**—So far the disease is only known in Jamaica and it must be remembered that the tree is a native of West Africa. We however do not know whether the fruit is used as a food

**ackee season** Several members of a family are taken ill at one time

**indigenous population** There is no indication that sex plays a part

**vomiting sickness** and as the experimental animals.

**Morbid Anatomy.**—There is a general hyperæmia, with a tendency to hæmorrhages in various organs. The mucous membrane of the stomach and the bowels is congested while the lumen of these organs may contain a dark slimy substance. There is fatty

hours.

The poison appears to be an irritant to the stomach, and to cause vomiting, which may rid the body of it, when the patient rapidly

symptoms occur, and there is a rapid recovery.

More usually however, after a period of temporary relief the vomiting commences again, and may be accompanied by fever, while the vomit consists of frothy mucus. These symptoms continue until the child passes into a state of collapse with cold sweats, a weak and rapid pulse and irregular respirations.

These symptoms invariably lead to death, which is preceded by convulsions.

**Variety.**— cerebral symptoms, cerebral death.

This is the

**Diagnosis.** re—Its endemicity, its prevalence, its sudden onset in members of one family or in neighbours, in native children without regard to sex; the quick complete recovery of some cases, while others, after showing cerebral symptoms end fatally, and finally the evidence of having partaken of a meal containing ackees or their extracts. It can be diagnosed from *yellow fever* by the absence of the black vomit, and from *cerebro-spinal meningitis* by an absence of Koenig's sign and of the meningococcus.

**Prognosis.**—This is very bad, as some 80 to 90 per cent. of the patients die. If recovery is to take place, it is rapid and complete.

**Treatment.**—No specific treatment is known.

**Prophylaxis.**—Instruct the people not to use unsound ackees. This has been done, with the result that in 1916 there were only three deaths from vomiting sickness in Jamaica.

## ONYALAI

in Angola  
 10 year in  
 st Africa

where it is called *edyuo* by the natives of Bukoba Mense (1906) thinks that the *lafindo* disease of the Unyamwezi people of the Congo is the same disease. Hæmorrhagic bullæ in the mouth but without general symptoms have been described by Maxwell in

thinks it may be some kind of poisoning perhaps with some species of the Euphorbiaceæ Wellman considers that it is not a manifestation of malaria nor is it a vegetal poisoning nor a snake poisoning though the bite of *Bitis arietans* the puff adder simulates some cases closely

and a dazed appearance. Sometimes the parotids are tender and the eyes may be somewhat reddened and in about 66 per cent of cases there is a slight rise of temperature. Numbness and pain in various parts of the body may be noted.

The appetite is poor. Bullæ may be seen on the tongue and in the mouth and pharynx while they also occur in the œsophagus stomach and bowels. The tongue is swollen and painful. Vomiting of blood is not rare and bloody diarrhœa may take place. Hæmaturia has been noted and cerebral hæmorrhage with the usual signs has been seen. It is believed that hæmorrhage into the pancreas liver and spleen may take place in some cases. Bullæ may also appear in the skin ranging from the size of a split pea to several inches in diameter. The typical bullæ whether on a mucosa or in the skin extend deeply involving the submucosa or the corium and are crossed by fibrous trabeculæ in the meshes of which lies partially coagulated blood which appears dark through the skin or mucosa. The red corpuscles are not disintegrated and can be seen by the microscope.

The disease is said to have a tendency to recur two or three times.

disease or peliosis is diagnosed by the rash painful swelling of the joints and the purpuric eruption. Henoch's purpura is met with generally in children and has joint symptoms as well as a rash

Hutch and *Albi sa anthelmintica* A Broga Massey recommends large doses of bicarbonate of soda and cod liver oil

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## CHAPTER LXXIII

### PELLAGRA

Synonyms — Etymology — Definition — History — Climatology — Etiology —  
Pathology — Morbid anatomy — Histopathology — Symptomatology —  
Diagnosis — Prognosis — Treatment — Prophylaxis — References.

**Etymology.**—The name 'pellagra' is an Italian word, possibly coined by the peasants from two words—*pelle* meaning 'skin' and *agra*, meaning 'rough'—though other possible origins are also given. It was introduced into medical literature by Frapolli in 1771 in his work, 'Anmadversiones in Morbum Vulgo Pelagram,' when he spelt it with only one *l* instead of with two.

**Definition.**—Pellagra is an endemic disease, usually of long duration and of unknown causation which is characterized by cutaneous, gastro-intestinal, and nervous symptoms, which undergo exacerbations at recurrent intervals usually in the spring or autumn.

**History.**—When it is considered that pellagra has been overlooked in quite recent years in America, Scotland, and England,

suffered, markedly resemble pellagra, as he had an eruption upon the hands and feet associated with an extraordinary mental condition.

by Columbus in 1497  
Milan as 'pellarella',  
erysipelas, and scurvy, and  
no real importance can be  
attached to a simple name  
unaccompanied by any de-  
scription of the disease to  
which it was applied. A  
slightly more definite refer-  
ence is found in 1713 in  
Ramazzini's work, 'De  
Morbis Artificum Diatriba'  
under the heading 'Agricola',  
where he says 'Las dem ob causas  
is persepe contingunt dolores  
colici et affecto Hippochondriaca  
quam ipsi appellant, il mal  
del Padrone'.

The recognition that the  
cutaneous gastro intestinal,  
and mental symptoms ex-  
hibited by the sufferers  
constituted a clinical entity  
was first made by Gaspar  
Casal on March 26, 1735,  
but, unfortunately, was not  
published until 1762, when  
his work 'Historia Natural  
y Médica del Principado de  
Asturias Sequida de la  
Descripcion de la Enfer-



de Pharmacie in May 1755. It is pleasing to record that not merely did Thiery give a most excellent description of the clinical picture of the disease, but also that he was the first to describe it in the town of Casal for his as yet

in the foundations of

only required an historian to become recognized by the medical community.

it under the charge of Gaetano Strambio whose justly celebrated work *De Pellagra* appeared in three volumes during the years 1786-1789. In 1787 two young Dutch doctors, Jensen and Hollen- hagen and a young Frenchman, Levacher de la Feutrie visited Italy to study pellagra concerning which they published reports on their return to Holland and France. In 1799 Chevalier gave an account of Jensen's work in the *London Medical Review and Magazine*. In this way the knowledge of the disease called pellagra started and spread.

It had been

not till Fanzago in 1789 published his work *Memoria sopra la Pellagra del Territorio Padovano* that these two diseases were

so forcibly by Casal and others. The importance of this work is

Balardini in 1815 in his work entitled *Della Pellagra del Grano turco quale Causa Precipua di quella Malattia e dei Mezzi per*

but the main subject was

has been promulgated that the disease is due to good maize which in certain people produces the symptoms of the disease and in others does not. Such briefly was the condition when Sambon in 1905 began his work which he has continued up to the present time. This work which is mainly epidemiological has thoroughly shaken if not completely destroyed the maize theory and has brought forward many facts which support strongly a parasitic

as the fact that Feijóo had recognized it in Galicia. The next publication is by an Englishman called Townsend entitled *A Journey through* in 1791. In account of *Pellagra in*

disease, and came to the conclusion that 'Mal del Hígado,' 'Mal de la Rosa,' and pellagra were one and the same disease. In 1835 Mendez Alvaro recognized pellagra in the malady called 'Flema Salada,' a  
 same disc  
 disease 17

of Oviedo

The maize theory of the ætiology of pellagra has never gained credence in Spain and hence the term 'Spanish pellagra, or a pellagra not due to maize' has been used by some Zeists as a medical synonym for a hoax

*Portugal*—Pellagra is known to exist in Portugal, but we are in the same condition as older writers, in that we can give no history of its recognition or spread

*France*—We have already noted Thierys publication in 1755 and the fact that in 1787 a young Frenchman, Levacher de la Feutrie, proceeded to Italy to study the disease concerning which he subsequently published accounts in 1802 and 1806, but it was Hameau in 1818 who first recognized the disease in France as occurring around Teste de-Buch in the Plain of Arcachon. Hameau was not acquainted with the literature which had sprung up concerning pellagra and his observations were in reality a rediscovery of the disease. In 1829 he read an excellent dissertation on the subject before the Society of Medicine of Bordeaux under the title 'Description d'une Maladie Nouvelle.' It would appear that he considered it to be an infection in some way acquired from sheep. In all he observed no less than seventy six cases.

There is however, evidence in favour of the suggestion that pellagra had long existed in France and this is to be found in the fact that the peasants used to call the disease 'Mal de Saint Amans,' because there was a statue to St Amans in Bascons which was always moist, and this moisture was used by the pellagrins as an application to their eruption. According to Roussel, there was another curious custom followed by the peasants of the Landes which was to visit a certain statue in which the Christ was represented with red hands. The priest was wont to apply an ointment to these hands from which the sufferers removed a little of the  
 face etc  
 'Mal des  
 l'Arouse

and 'Mal de Sainte Rosa' tend to show that the common people were well acquainted with the disorder. Sambon has also informed us that some of Napoleon's soldiers became affected by pellagra during the campaigns in Italy and certainly cases are recorded in the Hôtel Dieu and in the Hôpital Saint Louis while Jourdan published a paper on the disease in 1819



he had seen cases in Iceland resembling the pellagra which he had observed in Italy

*Africa*—Though first noticed in Egypt by Pruner in 1847 under

consider pellagra a part of the syndrome of ankylostomiasis. More recently pellagra has been recognized in other parts of Africa and it is possible that it will eventually be found to be widespread in that continent

*America*—Notwithstanding careful inquiries Wood has failed to discover any evidence of the existence of pellagra among the North American Indians during the eighteenth century but he has found some evidence of cases in the early years of the nineteenth century. In 1864 Gray and Tyler first definitely reported cases in them but though at times recognize

to be present until 1907

of which fifty seven were

cock Lavinder Siler Roberts Niles Wood and many others have made the disease well known. In the meanwhile pellagra has been

a  
g  
nd

*Summary*—The above history of pellagra demonstrates the ease with which the disease may be overlooked, even when abundantly present by well trained and most competent observers. It also impresses us with the fact that pellagra should be looked for in every country w

*Climatology*—

passed pellagra

At present it is known to exist in—

*Europe*—Scotland England France Spain Portugal Italy

South Africa

*Asia*—Asia Minor Persia India and the Straits Settlements

*Oceania*—Sandwich Islands

*America*—United States Mexico West Indies Brazil Columbia and the Argentine

dition *per se* is not so important as an epidemiological inquiry into the actual localities in which the disease occurs. Pellagra is essentially a disease of long duration and patients are capable

tracts and not in towns and that the densest localization is in houses near or alongside certain streams. In fact, study the locali

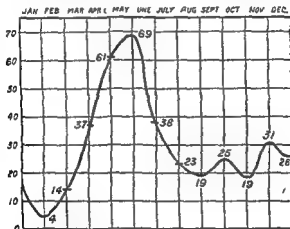


FIG 737—SEASONAL VARIATION IN THE ADMISSIONS FOR SIX YEARS OF PELLAGRA CASES INTO THE KASR EL AINY HOSPITAL CAIRO  
(Specially prepared by Keatinge and Shaven)

zation in what country you will as we have done in France Spain Italy Austria Hungary Roumania and Egypt it is impossible not to be impressed with the relationship between pellagra and water. Generally the water is moving and often it is moving rapidly but this last does not appear to be absolutely necessary. As a rule the nearer the dwellings are to such water the greater the number of cases. Cases do however occur at a distance from water but inquiry will often demonstrate that the affected people work near or have been in some way connected with a stream. Cases do occur in towns but they are relatively few, and careful inquiry will usually elicit a perhaps almost forgotten fact that the disease really began after some visit to the country. Inquiries however must be made with care otherwise wrong impressions may be obtained. One of the most interesting cases



which we have met with was that of a young boy who was supposed never to have left a large town and yet was suffering from pellagra. Careful inquiry elicited the fact that he was in the habit of going for a day or so every year to stay with some relatives who lived in a pellagrous area and the time of the year chosen for this visit was one in which acute cases occurred. In a locality pellagra usually occurs among the poor especially among field labourers, but it may also occur among the rich and among persons who habitually work indoors it is however usually not difficult to trace a relationship between the commencement of the disease and a visit or residence in some pellagrous area and very

common. In the autumn however there is a definite though secondary increase in the number of cases which decrease almost to nil in the winter. These statements are well borne out by the curve of pellagra admissions into the Kasr el Ainy Hospital Cairo for the years 1906-1911 inclusive. This curve for which we are indebted to the kindness of Dr Keatinge was most carefully prepared by Dr Steven and demonstrates the incidence of pellagra as seen in that hospital.

Pellagra may occur in hilly or even mountainous regions where it is often very common—as for example the Tyrol—but can

To summarize pellagra has a world wide distribution. It occurs in hills and plains. The cases are densest near moving

the present time  
sider possibilities  
instead of facts—a point which we desire the reader to bear in mind  
while perusing the remarks we are about to offer for his con-  
sideration

disease

2 That it is not a disease *per se* but merely a series of stray symptoms and that no one has studied the cases from the commencement of the disease

ankylostomiasis

The above can be easily dismissed and would not have been mentioned if we had not personally met with believers in all of them. With regard to the more likely theories we must discuss the following —

- I The deficiency theory
- II The maize theory
- III The parasite theory

I THE DEFICIENCY THEORY — In Chapter IV p 94 we have

whether pellagra may not be due to the lack of some nitrogenous

unanalyzed can be written in support of the maize theory in general. It can be pointed out that maize (*Zea mays* Linnæus) was originally a native of America where it has been found in its ancient form of small grains in the graves of the Incas and that it was introduced by Columbus or his followers into Europe where it did not grow

well in certain countries, like the British Islands, which, therefore remained free from importation of maize fore but where now children, and it may possibly be made into bread, cakes, scones porridge grain and tha

by the appearance of pellagra in that country, and quotation supporting maize can be drawn even from the writings of its most vigorous opponent, Sambon, who states — 'From authentic documents of the time we learn that "melica," or "fromentone" — i.e., maize—' was grown in Cremona in the sixteenth century and . . . in 1556 a Cremona nobleman offered the Duke of Florence ten *stata* of the new cereal' When this statement that there is plenty of maize in Cremona in 1556 — compared with the

taken by itself, would strongly support the maize theory is the well known fact that in the delta of the Nile there is plenty of pellagra

which statement must be received with caution as pellagra has so often been reported absent from places where it is now known, and, moreover pellagra is known to exist in Central Africa In Columbia pellagra is said to be found only in people who regularly take a drink made from fermented maize This drink is called 'chicha' and the malady 'chichismo' The Zeist states that pellagra is found wherever maize is used at all events, as an important article of food As a matter of fact, maize either growing or imported, is found all over the world, and pellagra would appear also to be found all over the world

care is taken, by inspection and by periodical chemical and microscopical examination, to exclude it A Zeist would answer to these objections that there was some fallacy and that nothing short of being the victim yourself would make it reasonably certain that maize was not consumed and even then it might have been eaten

insufficient foundations. It is however, necessary to lay before the reader the various phases of this theory, which may be classified as follows —

- 1 Photodynamic theory
- 2 Deficiency
- 3 Toxicity
- 4 Infectivity.

1. *Photodynamic Theory* — The special promoter of this theory is

might, under similar circumstances produce the same phenomena. This theory maintains that photodynamic substances are introduced by the cereals into the blood and these, under the influence of sun light become toxins, and thus cause inflammation of the skin

by excluding light from the skin of pellagrins by means of darkened rooms, red windows ointments bandages etc. Hirschfelder has searched for this fluorescent (photodynamic) substance in the blood serum of five patients suffering from severe pellagra and found that there was no difference in the fluorescence between their serum and that of healthy persons. Moreover sargopyrin only occurs in white animals and not in black, whereas pellagra can occur in the jet black negro which appears to us to be a strong objection.

2. *Deficiency* — This theory has been mentioned above with regard to the absence of nitrogenous complexes and it only remains to add that protein deficiency has also been brought forward as a possible explanation of the action of maize in producing pellagra.

have made investigations with regard to inoculating maize extracts into patients, obtaining several general reactions. These experiments support the latest view, which is really only the revival

of an older view that normal maize in certain individuals may produce pellagra or in other words that there is an individual susceptibility to maize. This subject has been recently carefully investigated by Rondoni in human beings. He procured his maize from the domestic store of certain pellagrins and having tested his cases for tuberculosis by von Pirquet's test administered extracts of the maize by intramuscular injections to thirty three pellagrins and thirty non pellagrins. He did not find any violent reaction as described by other writers but he found that recent cases of pellagra and convalescents reacted more definitely than non pellagrins showing slight fever headache malaise excitability and sleeplessness and considered that this increased sensibility might be regarded as an anaphylactic reaction to some undefined factor in the maize extract. If this anaphylactic theory held then

The third theory asserts that poisons are generated in the bowel from the grain (Neusser) by the aid of the *Bacillus coli communis* (De Giaxa). This latter theory of De Giaxa is supported by experiments for he produced the symptoms in animals inoculated by the toxin produced by growing the *B. coli communis* in maize media.

Numerous observers have reported poisons in fermenting maize. Thus Lombroso in 1871 obtained two alkaloids one like conium and the other like strychnine but the symptoms produced by these on men and animals were not like those of pellagra. Others have reported tetanic or narcotic poisons etc. but on the other hand Monselice failed to obtain any such poisons in damaged grain from pellagrous districts.

4 *Infectivity*—At the present time the popular belief is that the

Hirsch who points out that a bad maize harvest is followed by an increase in the cases of the disease. The theories as to the substance in the damaged maize which causes the disease are manifold and may be classed into (a) fungi (b) bacteria (c) chemical substances.

(a) *Fungi*—Monti and Tirelli showed that fungi were commonly found in maize those most usually met with being *Penicillium glaucum*, *Rhizopus nigricans*, *Mucor racemosus* and species of

um  
ise  
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on men and animals are quite different from pellagra. *Aspergillus fumigatus* and *Aspergillus flavescens* (or *A. varians*) have been obtained by Ceni in pure cultures from the lungs, pleura, and

of the disease

Tizzoni has described a bacillus found in the cerebro spinal fluid of pellagra patients and on maize, but this bacillus will be considered at greater length below

The maize theory is therefore by no means proved, and in fact is in our opinion, very doubtful

**Parasitic Theory.**—The parasitic theory of the origin of pellagra, which is supported, to a certain extent, by our own observations and by the Illinois Commission, who conclude that it is a disease due to infection with some living organism, may be classified into —

A *Vegetal parasite*

Tizzoni's streptobacillus

B *Animal parasites*

Alessandrini's theory, 1910

Long's theory, 1910

Perroncito's theory, 1910

Babès' theory, 1911

Sambon's theory, 1905

**TIZZONI'S STREPTOBACILLUS** —This is really a part of the maize theory, as Tizzoni has found the bacillus on maize, but it is also

family Filaridæ he considers to be the cause of the disease. He also states that he has found a filarial egg in the skin of pellagrins.

According to Sambon who has seen Alessandrini's specimen the thick shelled egg belongs to one of the nematode worms infecting pigs. Alessandrini's theory has not received much support up to date.

**LONG'S THEORY**—This theory suggests that pellagra is merely a phase of amœbic dysentery but the Illinois Commission as well as the observations of Sambon and one of us do not support this theory. Dysenteric like ulcers can be found in the intestine but they often do not contain amœbæ.

resembling a Chlamydozoon in the skin.

**SAMBON'S THEORY**—This theory is double-barrelled—it brings forward the proposition that pellagra is a protozoan infection and that it is spread by the agency of a biting fly.

**Parasite**—His reasons for believing that it is a parasitic disease are almost entirely epidemiological. They are—

1. *The Mononucleosis present in the blood*

2. *The Presence of Long Intervals of Quiescence followed by a Relapse*—Thus a young pellagrino with marked symptoms who comes to London and receives most excellent food without any admixture of maize suffers from a mild relapse every year in the month of April. In this case there can be no question of maize causing these relapses. Further we may state that the young

about sixty insane persons in each. One cottage was placed on a generous maize diet and the other on a maize-free diet. At the end of twelve months the maize eaters had four certain cases and one doubtful.

five certain

were merely

but as we ha

mission Report we cannot give details.

A point has been made that while patients in asylums develop acute symptoms the attendants do not and this is held to be an argument against the parasitic cause of the disease. But this is just where the experience of Sambon and one of us differs from those of many authorities because we have seen the disease begin so often in early childhood. The youngest case seen was three months of age and we have obtained excellent histories of long intervals occurring between one series of acute attacks and the next. We look upon these asylum cases as probably relapses of earlier

attacks and are therefore not surprised that attendants are not attacked because probably the agent of the conveyance of the disease is not present in the vicinity of the asylum

In other asylums however it is possible that if suitable conditions were present the disease might spread among the inmates and attendants

3 *The Constant and Characteristic Topographical Distribution*—We have already drawn attention to the topographical distribution and to the fact that pellagra remains endemic in the same localities for very long periods and we have also referred to the case of a young boy developing pellagra though constantly residing in a town after a brief visit to a pellagrous region. The case of the fishermen of Burano (*vide infra*) may also be quoted and finally attention may be invited to many similar instances quoted by Sambon in his able Progress Report on the Investigation of Pellagra published in London in 1910. Associated with this it may be mentioned that of two places almost contiguous one may be affected and the other not

4 *Its symptoms course duration morbid anatomy as well as therapy are similar to those found in parasitic diseases*

5 *Spirochæte*—Sambon found a spirochæte in the liquor from a bulla on the hand of a case of acute pellagrous dermatitis in Roumania

r Pathology and need not

to Sambon's theory is that definitely associated with time rectified in the near

future

The very few experiments performed on white rabbits by injecting liquor cerebro-spinalis blood from the erythema or lymph from bullæ subdermally or into the spinal canal have produced no definite results nor have attempts at intradermal inoculation of these animals with the same fluids been more successful. Neither have the attempts made by the Illinois Commission to transmit the disease to monkeys and guinea pigs met with more success. Recently however Siler in a communication to Sambon has stated

*living organism*

*Carrier*—Sambon however has not been content to remain with an incomplete theory but has advanced the view that the



3 It has a definite *seasonal incidence*—spring and autumn—which coincides with the appearance of certain flies

4 It largely affects *field labourers* and new residents in endemic areas

5 It is *not contagious* and neither food nor water can account for its peculiar epidemiology

6 In the endemic centres it *affects all ages* both sexes (as a rule females are more frequently attacked than males)

7 An endemic centre is one in which it is usually easy to find *young children* with the symptoms of the disease -

8 In endemic centres *whole families* may show signs of the disease but outside these only one or two individuals may be affected

*Researches on the Island of Burano in the Venetian Lagoon*—With regard to the theory of a biting fly Sambon is supported by the inquiry into the pellagra of the Island of Burano made by himself Colonel Belli and one of us in which it was found that the fishermen and the boys who went fishing with them were attacked by

of these young children girls and even women were alleged never to have moved from Burano with the exception in some instances of an occasional visit to Venice. These points are con-

pellagra. They are against sexual infection as the women would acquire the disease they are against infection by contact by <sup>ce but</sup> <sup>1 para</sup> <sup>rasites</sup> such as bugs or fleas or personal parasites such as lice. The

*terra firma*

a history

early morning or late evening

Sambon considers that the peculiar feature of the erythema coming in the spring and the autumn must be a correlation with

ere is

m the

it had two seasons, during which it appeared in swarms and attacked man and animals—viz., spring and autumn, and not in

ity is however, very strong

**Summary.**—It appears to us that while at present the causation of pellagra is unknown, and while the modern tendency is to claim it as a deficiency disease, still the investigations of a possible protozoan parasite and its carrier should not be given up

**Predisposing Causes.**—Sex would appear to be a predisposing cause, because the disease is often more prevalent in women than

underground in mines, and the women presumably in and about the houses, which were on the banks of a fly infested stream. Here the children also were much affected. In interesting contradiction is the incidence in the women of Burano, who mostly work indoors and among whom pellagra was very rare, but it was common among the fishermen and boys who fish in the rivers, etc., where biting flies are common

Age would not appear to have any marked influence, but it would seem as though the disease was very prevalent—in a mild form at all events—in the early years of life, as the children of a pellagrous district are often early affected, and some of these attacks are by no means mild but very severe

With regard to social position poverty, lack of sufficient food, and bad hygienic surroundings it was long considered that these had a marked influence in producing the disease, but though they may help, as they would, with almost any form of disease, still, the American and our own experience show that they have no real connection with pellagra which can equally well occur among the well-to-do, the well fed and the

RESISTANCE OF  
of pellagra  
enteric fever,

Pathology—As the causation of pellagra is entirely unknown

which can be produced in white animals fed on maize and exposed to sunlight and believe that these changes support the photodynamic theory

of the mononucleosis of the perivascular infiltration, and of the nerve cells as well as the long intervals of apparent quiescence as phenomena similar to those seen in the

and more rarely on the genital organs in the spring and autumn are brought about by the combined action of the parasite whatever it may be and sunlight in correlation with the habits of some biting fly. From epidemiological studies he suggested that some member or members of the family Simuliidæ might be the insect in question. He came to this conclusion because of the known habits life-history etc. of the Simuliidæ which were in general agreement with the epidemiology of pellagra.

The reason why there is such a confusion of ideas with regard to the pathology is not difficult to understand as a post mortem made on a case of acute pellagra within an hour or so of death is of great rarity and has often been performed under conditions of difficulty as regards cleanliness or preparation. Post mortems on cases of recurrences or of chronic pellagra have been abundantly performed under the best auspices but the main features of the disease are often obscured by secondary changes while even in the acute cases the phenomena are complicated by the presence of malaria typhoid tuberculosis syphilis etc.

Notwithstanding all these objections there is some evidence in favour of an early lesion of the central nervous system especially the posterior portion of the spinal cord in the lower cervical and dorsal regions as congestion and hæmorrhages have been found there while in the more chronic condition degeneration of the cells in the posterior cornu in Clarke's column and in the spinal ganglia have been seen as well as the later degeneration of some

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to sclerosis. Degenerative changes have also been recorded in

Purkinje's cells, in the cerebellum, and in the cortical cerebral cells

If these accounts are confirmed, they might be found to stand in relationship to the angio neurotic process in the skin the congestion of the alimentary canal the vertigo, and the mental condition

However, all these points are at present extremely obscure, and we only brought forward here because they appear to require

system and the autonomic nervous system

**Morbid Anatomy.**—The principal point in studying the morbid

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hemorrhages the ileum may be thinned and Peyer's patches may be

nerve roots may be implicated and the intervertebral foramina may be

intestinal lesions described above are met with in many post mortems in

nucleus and nucleolus in acute cases. Associated with this there is infiltration of the interstitial tissue and proliferation of the endothelial cells of the capillaries and circumscribed hemorrhages.

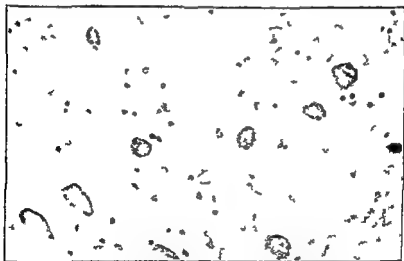


FIG 738—CELLS OF CLARKE'S COLUMN IN A CASE OF ACUTE PELLAGRA  
( $\times 50$ ) (After Sambon and Chalmers)

nerves

In the brain the cortical cells have been found to be degenerated, swollen and even disintegrated, while Purkinje's cells in the cerebellum are also said to degenerate. Mott has shown that while the fibrils surrounding these cells

may be intact those passing through the cells disappear. A perivascular inflammation

The cerebro spinal fluid is usually present in considerable quantity and

**Symptomatology.**—The incubation period of pellagra is unknown but it cannot be of long duration as we have known it to occur in a child three months of age and as we have already advanced arguments against heredity, this case is in favour of a short incubation period. We have also met with a case in which the skin symptoms are said to have appeared about two weeks after return from a

weeks

The description of a typical case is something of this nature — A person male or female young or old in apparently excellent health or in bad health living or working in the sunshine of a spring day notices that a sunburn appears on the backs of his hands and perhaps the dorsa of his feet if bare and more rarely also on his face or neck. He thinks little of it though the inflamed area burns and may even blister. Perhaps he has a sore mouth and perhaps he has a little diarrhœa or constipation. Perhaps he feels a little giddy in the morning and perhaps he is easily tired. In a week or so the redness dies down and the affected area is seen to be pigmented and perhaps to have the skin thickened in places. In the course of a few days or a week or so these thickened areas desquamate and the skin underneath

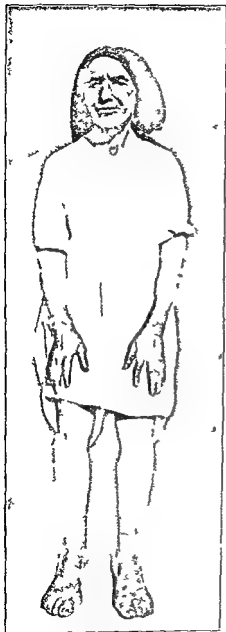


FIG 739—ACUTE EXACERBATION OF PELLAGRA

Note the marked erythema on the hands and feet and the less evident erythema on the neck and face

may be found to be normal or may be found to be slightly atrophic and to appear whiter than the surrounding pigmented area'

The patient thinks no more of his troubles the autumn comes and he feels well and during the winter there is no alteration in his good health. Perhaps the next spring may pass without a recurrence and perhaps even several springs may elapse before the patient who all this time may consider himself to be in excellent health has a return of his symptoms. But though the interval may be long or short this relapse will surely occur and often it will be in a severer form than it was at first. It will usually recur in the spring but it may take place in the autumn or much more rarely in our experience in the summer.

This time the symptoms may be mild as before but on the other hand they may be severe the erythema appears with severe burning sensations and a real dermatitis with bulla formation may develop.

The tongue lips mucosa of the mouth and palate may become inflamed and show the presence of vesicles and ulcers. The parotid gland may enlarge (this is common in Egypt but rare in other countries) the saliva may be so increased in amount that it pours from the mouth. There may be signs of dyspepsia pains in the abdomen and diarrhoea or even dysentery may develop. The

may become quite distressing to the patient who upon quickly rising from a sitting or a lying posture may even fall to the ground. The muscular power may now be diminished and the patient may no longer be able to do his work, tremblings in various parts of the body but more especially in the head and arms may be noticed and the legs seem scarcely able to support the

Again as winter approaches the symptoms will diminish and die away and the patient will feel better, but this time the skin does not recover itself but remains thickened and pigmented in places and thinned whitened and atrophic in other places.

Again there may be only an interval till the next spring or autumn or the succeeding spring or there may be a longer interval and again mild or severe symptoms may appear.

With repeated attacks the skin changes become marked the mind becomes often permanently affected and melancholic or

noted especially in the legs. The knee-jerk is at first exaggerated but later is diminished and finally lost. Ankle clonus and wrist clonus are rare and only occur when the knee jerk is exaggerated when there is tenderness on both sides of the dorsal and lumbar

falling down. There may be tremors in the legs and tongue. The bladder and rectum are not as a rule affected until the end but there are exceptions to this and contraction of the limbs does not take place till bedridden but moderate rigidity of the muscles of the arms and legs associated with stiff and at times irregular spasmodic movements has been recorded as occurring early in rare acute cases. A sudden rigidity of the body associated with re-



is the history given by the friends of the development of some extraordinary habit of diet to the ...  
is often assign  
be due to the  
in pellagrins

We have rather insisted upon the occurrence of pellagra in young children because we are of the opinion that it is often overlooked

*Skin Eruption*—The skin eruption is usually limited to the regions exposed to the sun's rays

the hands and feet, only reaching the flexor aspect after several attacks. The most common sites are the hands and forearms, elbows, feet, legs and knees, the upper part of the chest, the shoulders, neck, and face. More rarely it appears on the genital organs and around the anus in both sexes. Sandwith points out that in Egyptian peasants the unguinal phalanx of the hand is seldom affected, due to the fact that when the hoe is grasped it is protected against the sun's rays. The nails and hair are not affected, but rarely there is a dry, scaly condition of the palms of the hands.

The skin eruption may show the following conditions—(1) Congestion, (2) inflammation, (3) thickening and pigmentation, (4) atrophic thinning

lasting a variable period of days or weeks, the eruption gradually subsides, leaving the skin rough, pigmented and thickened, and thus earning the names of 'pellagra' and 'qushuf'

This erythema disappears in the winter, but reappears the next spring with increased virulence, and in due course develops into a dermatitis, which produces an exfoliation of the epidermis in grey or brown flakes. Every attack leaves the affected area a little

on the back, hands, and feet. Special terms have been given to the eruption when in certain areas—e.g., the 'glove,' the 'boot,' Casali's 'necklace,' or 'cravat,' and the 'mask'

Roberts has introduced the rather useful term 'dermotagra' for the dry, scaly, thickened skin seen over the olecranon process, over the knees, and more rarely on the palms of the hands. Over the elbow and knee the skin is rough, thickened, and wrinkled; on the palms of the hands it is merely rough and thickened

Another interesting dermatological feature of pellagra is the

frequency of nasal or facial hemorrhage, which is to be especially very prominent

with a whitish fur during the onset of an attack, but later it becomes abnormally clean—'Sandwich's bald tongue'—red swollen, and sometimes ulcerated near the tip. It may also become fissured, but in mild

, and may bleed the palate, as well as rhinitis, and more

in number, and the like form of the red

heart rate may be markedly increased; vasodilation of the extremities, the bluish congestive paralytic or an appear-

is not affected here may be an acute stages of

or nearly so, and any great change must be considered to be a complication

*Sexual Organs*—Sexual power is usually diminished, especially in the later stages. Amenorrhoea, metrorrhagia and inflammatory conditions of the vagina, uterus, ovaries, etc., are described, but

is changes dependent produces

is normal 101° to may also

with long duration of infections,

may give apparently

simply a pellagrin with an infection with one or more of the

examination immediately after death

but the frame of mind in the observer in attempting to write upon this subject

For the diagnosis of pellagra two conditions are necessary in the observer. The first is that he must suspect its presence and be on the outlook for it in any and every country and the second is that he must not be unduly swayed by any ætiological theory and must be prepared to make a diagnosis of pellagra in a person of any age any race any social condition living in any place whether tropical temperate or frigid resident in a town or in the country and he must do this without consideration of the dietary or the surroundings with perhaps the sole exception of being more intently awake to the possible occurrence of the disease in lunatic asylums.

As there are at present no microscopical bacteriological parasitological hæmatological or chemical reactions which can be said to be diagnostic of the disease with perhaps the sole exception of the pellagra like symptoms produced by one of the American

festations.

The cardinal signs of the disease may be summarized into—  
(1) *the cutaneous signs* (2) *the gastro intestinal signs* and (3) *the nervous signs*

In order to make a definite diagnosis there must be either the presence or a definite history of the cutaneous signs of pellagra  
 her groups  
 or less sym  
 ondition more  
 backs of the

hands the dorsa of the feet the face the back and sides of the neck or the front of the chest especially if these eruptions are limited by a more or less definite elevated margin to the areas habitually exposed to light suspicion should at once be aroused

absent. Those most commonly met with during exacerbations are—Salivation stomatitis dyspepsia due to hypochlorhydria diarrhoea laryngeal symptoms alternating with constipation or simply constipation appearing or recurring in the spring or autumn. A diagnosis cannot be made by these symptoms alone which must be considered in conjunction with the cutaneous in order to arrive at a conclusion. If no cutaneous symptoms are visible it is justifiable to place the patient in strong sunlight in order to see whether the dermatitis will appear.

3 *Nervous Symptoms*—Of all the nervous symptoms early ex-

condition of the temper recurring in the spring or autumn but these must be associated with evidence of skin lesions before a diagnosis can be made.

rarely from attacks of mania. It may be noted that at times he

means that he has an excess of saliva but much more importantly it is noticed that he suffers from *chronic eczema* on the back of his neck often a view really

pellagra

■ *Young Children*—In young children the disease is very apt to be overlooked.

ie ions. A careful inquiry will show whether these symptoms have or have not a seasonal incidence and in any case they should arouse suspicion of pellagra which should only be eliminated after careful inquiry.

Differential Diagnosis — As the symptoms are divisible into

appearances.

pellagrous eruption at times appears on parts not usually exposed to the sun and by the fact that the lesions in sunburn are usually very superficial

involvement of

From *acarine* the  
localization of the  
by the absence unk  
absence of itching r the

From *alcoholic erythematata* it may be recognized by the history of the attack and by the presence of the typical eruption on the hands and feet

Some authorities have however described under the term *pseudo pellagra* of alcoholic origin an erythematous eruption on

skin as well as by the more regular distribution of the eruption and by the limiting line

2 *Resembling the Chronic Dermatitis*—From *chronic eczema* occurring in mentally sound persons or in lunatics pellagra is recognized by the typical distribution of the eruption by the line of demarcation and by the marked pigmentation when present and absence of pruritus. From *chronic syphilides* by the distribution of the eruption and by the absence of any reaction to mercury. Here mention may again be made of the *dermatagra* on the palms of the hands over the olecranon and about the knee in chronic cases of pellagra which is apt to be overlooked or to be considered as points in favour of a diagnosis of eczema rather than of pellagra.

From *biotripsis* (*vide p 2282*) which it closely resembles pellagra is differentiated by the limitation of the eruption to the areas so often

The wrinkled skin of the washerwoman's fingers is hardly likely to be confused with the chronic thickening found in pellagra,

lesions but here care must be taken not to mistake the dermal signs of pellagra for chronic eczema, etc., but this point, having been already discussed above, need not again be argued.

**Prognosis.**—This would appear to be good in early cases in mild cases, and even in moderately severe cases if the patient can be removed from the pellagrous area and placed in good condition of

guarded and it must be after two five and even

In severe cases the prognosis must be guarded and the low blood-pressure remembered as well as the possibility of sudden death from exertion after lying down.

Cases of typho-pellagra have a high mortality, and here the prognosis is obviously bad.

Complication with ankylostomiasis, tuberculosis, etc., also

render the prognosis more unfavourable as does continued residence in a pellagrous area

**Treatment**—There can be no doubt that the essential basis of the treatment of pellagra is to remove the patient from the pellagrous area in which he has been living to a non pellagrous area and secondly to give a good and liberal diet preferably without any admixture of maize although Devoto has shown that good maize not merely does no harm to pellagrins but is very suitable for some of them as it is their usual diet. If this is done most early cases quickly improve and apparently are cured but unfortunately this is not so because even if they remain under these excellent conditions sooner or later a recrudescence occurs. These recrudescences may be mild but at any time they may become severe even when maize is excluded from the dietary.

**Arsenic**—This being so it is obvious that some medicinal treatment is necessary in addition to change of locality and diet and apparently the best remedy is arsenic in some form. Of all forms of the drug that most commonly in use and also much vaunted is 'atoxyl' which is administered by intramuscular injection of 3 grains per diem.

Other methods of giving arsenic are salvarsan neosalvarsan and soamin.

Other methods are the racodviate of sodium administered in 3 grain doses by intramuscular injection every third day until three doses have been given and then every second day until three more doses have been administered and then increasing to 5 grain doses every second day until the symptoms have improved.

Associated with these injections it is as well to give liquor arsenicis in small doses internally and to continue this interruptedly for some three months after the symptoms have disappeared. In addition it is as well to repeat the liquor arsenicis some weeks before the advent of spring and to continue it intermittently into the summer for a few years after an attack in order to attempt to guard against the almost inevitable relapse.

in the cases resulted

**Symptomatic Treatment**—The patients must be protected from the sunlight by clothing veils hats gloves etc and the derma  
calmure soothing

containing dilute

located on

With regard to the nervous symptoms the irritation on retiring to bed and the sleeplessness should be remembered and combated with cool bathing and when necessary by doses of bromides which however are apt to increase the depression

**Diet.**—The diet must vary with the condition of the digestion and the bowels and during attacks of dysentery or diarrœa that laid down on p. 1858 should be adopted.

**Complications.**—Search should be made for signs of ankylostomiasis ascariasis etc tuberculosis malaria etc and these should

as above

**Prophylaxis.**—As the ætiology of pellagra is unknown it is

accurate they may result in much misconception of the incidence of the disease. The provision of one good meal a day to poor pellagrins during the spring and autumn is in our opinion highly to be praised. That these meals are good we can certify from personal experience. We fail however to see the utility of the free distribution of salt but it does no harm

The methods adopted by the Italian and other Governments may be summarized as follows—

#### 1. *Laws and Regulations*

- (a) Prohibiting the importation and sale of spoiled corn
- (b) Government inspection of all corn stored or consumed. This includes the erection of public storehouses
- (c) Provision of dehusking plants to dry corn



(d) Cases of pellagra to be reported, and lists to be kept and emended

(f) Free distribution of 17½ pounds of salt to every adult pellagrin and

(i) Financial supply

### 2. Formation of Rural Bakeries

Model central bakehouse, controlled by Government, in which the only bread allowed to be used is baked from good, wholesome wheat flour. The best model we have seen was in the Tyrol

### 3. Improvement of Agriculture.

(a) By *calledre ambulanti*, or farmers' institutes, designed to teach locally modern methods of agriculture, with the result that the farmers become less

often only half ripe, and soon decayed

(c) By organizing *agricultural shows*, which include the exhibition of *maiss*

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## **SECTION C**

### **SYSTEMIC DISEASES**

**DISEASES OF THE ALIMENTARY CANAL.**

**DISEASES OF THE SYSTEMS**

**SKIN DISEASES**

**DIVISION I DISEASES OF THE ALIMENTARY CANAL**

**DISEASES OF THE MOUTH AND STOMACH**

**HELMINTH INFECTIONS**

**SPRUE AND THE DIARRHOEAS**

**THE CHOLERAS**

**THE DYSENTERIES**

**INTESTINAL SCHISTOSOMIASIS**

**EPIDEMIC GANGRENOUS RECTITIS**

## CHAPTER LXXIV

# DISEASES OF THE MOUTH, THROAT, AND STOMACH

General remarks—Oral infections—Thrush—Gingivitis—Lingual affections—  
ifalzoun—Tonsillar affections—Gastric diseases—Earth-eating—Bel  
yando spew—Entalacão—References

### GENERAL REMARKS.

only met with one example and then of a most unusual form the

regions but we have seen primary tuberculosis. *Appendicitis* is quite common in both Europeans and natives, and is caused by bacteria acting either directly, or introduced by the action of *Trichuris* and *Ascaris*. *Appendicitis* of schistosome origin has been recorded by Mursell.

In previous editions we called attention to the frequency of *intestinal sand*. The true intestinal sand of animal origin composed of small particles of sand and largely composed of more common is the undigested remains of *secum* encrusted with the condition 'facal sarcoma.' In these cases there may be diarrhoea and colicky pains.

*Uronema caudatum* has been recorded by Fischer in 1914 in the diarrhoeic stool of a European in Shanghai. Two days later only cysts were present.

### ORAL INFECTIONS.

The protozoal parasites reported as being found in the mouth are —

*Loeschia gingivalis* Gros.

*Leishmania tropica* Wright var *americana* Laveran and Nattan Larrier

*Spiroschaudinnsadentium* Miller, and many other spirochætes

*Spiroschaudinnsia buccalis* Steinberg

*Treponema mucosum* Noguchi

*Treponema macrodentium* Noguchi

*Treponema macrodentium* Noguchi

Flagellates have also been recorded

*Gongylonema pulchrum* — This filarial worm is a parasite of the pig in Europe and America but was described in 1916 by Ward as

The most common and perhaps most serious affection is *Pyorræa*

amoebæ spirochætes bacteria etc.—and therefore the causal germ is unknown but the treatment is quite clear—viz to remove all teeth which are too far decayed to allow any hope of improvement or which are viewed as dangerous from a general health point of view. Having done this the next and most important point is *ionisation* with zinc sulphate.

It will be remembered that streptococci are apt to enter the system

and streptococcal are common in the tropics though diphtheria is somewhat rarer than in temperate climates and require careful treatment with local antiseptics and either serums or vaccines.

*Salivary calculi* have been reported by Christopherson and are certainly not uncommon in Europeans and natives and are apt to

recur after removal. A favourite seat appears to be the sublingual duct where the calculus is apt on superficial examination to be

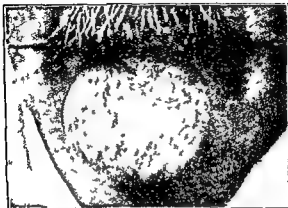


FIG 74 — LEUCOPLAKIA

*Leucoplakia* and cancers of the lips, cheeks and tongue are frequently met with in the tropics and appear in a curious way to be related to mycetoma via the condition called *paramycetoma* (vide Chapter XCIII, p 2145)

### THRUSH

**Synonyms** — Saccharomycetic stomatitis. Oral oidiomycosis. French *Muguet*. Millet *blanchet*. Italian *Mughetto*. German *Schwimchen*.

**Definition**.—A stomatitis or more correctly a group of stomatites characterized by the presence of creamy white patches believed in the past to be produced by *Oidium albicans* Robin while at the present time it is recognized that they may be produced by a multiplicity of fungi.

**Historical**.—The affection was clinically known to Hippocrates forming part of the *σροματις ἀφθώδης* described by him. It was also certainly known to Galen under the term of *aphtha a'ho infantis*.

Suivege called the affection *aphtha lactantium* and Bateman *aphtha lactantium*.

might be the cause of this malady



**Diagnosis**—The diagnosis can often be made clinically the creamy white patches being characteristic, but it should always be confirmed by the microscopical examination which will reveal a large amount of mycelial threads and conidial forms. If it is thrush the patient is These are described

**Prognosis**—Thrush *per se* is not a serious affection but its occurrence in cachectic patients is a bad omen

**Treatment**—Glycerine of borax applied to the patches several times a day is efficacious in many cases or an aqueous solution of borax (1 in 30) may be used. The addition of honey to the latter is to be deprecated. In resistant cases the addition of carbolic acid to the glycerine of borax 10 minims to the ounce will be found useful. In marasmic children or adults suffering from some incurable disease any treatment may fail to bring about a complete disappearance of the thrush.

**Prophylaxis**—In the case of infants there is no doubt that in many cases the infection is carried by contaminated nursing bottles and their rubber nipples. These should therefore be kept scrupulously clean a number of thrush fungi; the use of alkaline tooth pastes and mouth washes to be recommended

## GINGIVITIS

During the last few years much attention has been paid to the gums which may be divided into localis and ulcerative gingivitis

### Simple Gingivitis

In this disease the gums are bright red in colour especially near the margin becoming normal when traced towards the buccal mucosa. The interdental papillæ are swollen but are neither painful nor ulcerated. There is no odour no pain at night and no enlargement of lymph glands but the teeth may be covered with tartar and show food debris. A carbolic rose water mouth wash ( $\frac{1}{2}$  per cent) will be found useful

### Pyorrhœa Alveolaris

### Ulcerative Gingivitis.

ulcerative stomatitis, and a membrano-ulcerative gingivitis, two of which are commonly present when a case comes to be noted, although the gingivitis is nearly always the primal disease.

All forms are common in the tropics, and their relative frequency has been investigated in Palestine by Schimeoni Meckler, in 1917, who found that 78 cases of mouth disease could be resolved into 28 cases of ulcerative gingivitis, 17 of ulcerative stomatitis 6 of Vincent's angina and 27 of mixed types. The whole subject has been ably studied by Barlow in 1914, Bowman in 1916 by Taylor and M. Kinsty in 1917, and by Colyer in 1918. There are three varieties of the complaint—viz., the acute, the subacute, and the chronic.

**Acute Variety.**—This is an acute inflammation of the margins of the gums, of gradual onset, but which spreads rapidly and causes ulceration of the interdental papillæ and sloughing of the gums around the necks of the teeth and in severe cases ulceration of the oral mucosa associated with malaise, fever, and enlargement of the lymph glands, hemorrhage from the gums, and pain therein, especially at night, bad taste in the mouth, offensive breath, difficult and painful mastication, and loose and tender teeth. There is

commonly met with in persons living under bad conditions.

**Subacute Variety.**—The gums are spongy and tender, and a whitish pellicle often forms which on superficial examination may give the appearance of a purulent exudate. The condition often spreads

pass into the chronic, and lead in a year or so to destruction of the bony sockets.

**Treatment.**—The best treatment for these infections is to remove the tartar, disinfect the mouth with a spray of peroxide of hydrogen or of glycothymoline and then to treat by ionization with zinc sulphate, and afterwards to use anti-septic washes of sintas or similar preparations. Roberts recommends the local application of the following: Hydrogen peroxide  $\mathfrak{v}$ , vinum ipecac  $\mathfrak{m}$ , glycerin  $\mathfrak{v}$ , aq ad  $\mathfrak{viii}$ .

## LINGUAL AFFECTIONS.

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*Lingua nigra* is occasionally seen. We have already called attention to the *dark patches* found on the tongue in natives, and which by some writers have been described as a sign of ankylostomiasis. These pigmented patches are roundish or oval, and may be found also on the gums, the mucosa of the lips, on the soft and hard palate, and are apparently congenital. A condition which might be called *Red or Purple tongue*, and which often puzzles the newly arrived medical man who does not know its origin, is extremely common in Ceylon among the coolies and lower-class natives, and is simply due to chewing betel. The pigmentation slowly disappears on the native discontinuing the use of betel. Cases of *Furrowed tongue* (scrotal tongue) are not rare. We have seen a case of *Fordyce's disease* (pseudo colloid of the lips) in a half caste. A case of *Chelitis exfoliativa* in a European lady and cases of *Perlèche* have been observed by us among European children. Under the term *seasonal recurrent ulceration of the lips*, Gros has described a very superficial ulceration on the lower lips in Algerian natives which is very common in the hot season, and is due, according to him, to a diplobacillus.

*Pityriasis Linguae Spirochætica*.—This condition has been described by Castellani. There is as a rule no sign of acute inflammation and no ulcers, but the dorsum of the tongue is covered by a thick, persistent, whitish-yellowish or greyish brownish fur, which on microscopical examination seems to consist solely of innumerable spirochætes, with some epithelial cells. Of course, a few spirochætes are always found in scrapings from the tongue, but never in such enormous amounts.

## HALZOUN.

Definition.—Halzoun is the invasion of the pharynx by the

in Northern Lebanon

Ætiology.—The disease is caused by eating raw livers, especially raw goat (*Capra hircus* Linnaeus) livers, which are infected with

gia,  
but  
tim

## TONSILLAR AFFECTIONS

Every type of tonsillitis met with in temperate climates is observed also in the tropics although there is no doubt that tonsillar and throat affections are less frequent in warm climates than in cold. Diph-

association very often with spirochetes. We do not propose giving a description of Vincent's angina which may be found in any text book on general medicine but we would call attention to the possibility of mistaking it for a syphilitic condition. Certain authorities state that Wassermann reaction is positive in Vincent's angina but in our experience this is not so and we can confirm the researches of Taylor and others according to which Wassermann reaction is negative in Vincent's angina except of course when it develops in a syphilitic person. Cases of tonsillitis probably due to amebae and flagellates have also been recorded. We propose saying a few words on certain affections of the tonsils to which little attention has so far been paid—viz. mycotic infections.



FIG 743.—TONSILLAR AFFECTION CAUSED BY *Hemyspora rugosa* CASTELLANI.

Tonsillar nocardiomycosis and lesions of the tonsils due to *Nocardia bovis* and other species of the genus *Nocardia* and

consult the doctor because of sore throat but because of the unpleasant odour of the breath. On examination the teeth and gums may be quite healthy but on examining the throat small whitish

## LINGUAL AFFECTIONS.

Patches may be of various colors and are so called because they are rare. Little attention is paid to the *dark patches* found on the tongue in natives and which by some writers have been described as a sign of ankylostomiasis. These pigmented patches are roundish or oval and may be found on the lips on the soft palate and on the uvula. A condition which puzzles the newly arrived medical man who does not know its origin is extremely common in Ceylon among the coolies and lower class natives and is simply due to chewing betel. The pigmentation slowly disappears on the native discontinuing the use of betel. Cases of **Furrowed tongue** (scrotal tongue) are not rare. We have seen a case of **Fordyce's disease** (pseudo colloid of the lips) in a half caste. A case of **Chelitis exfoliativa** in a European lady and cases of **Perleche** have been observed by us among European children. Under the term *seasonal recurrent ulceration of the lips* Gros has described a very superficial ulceration on the lower lips in Algerian natives which is very common in the hot season and is due according to him to a diplobacillus.

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**Definition**—Halzoun is the invasion of the pharynx by the

in Northern Lebanon

**Ætiology.**—The disease is caused by eating raw livers especially raw goat (*Capra hircus* Linnæus) livers which are infected with *Iasciola hepatica* (Linnæus 1758) when the worm (p. 565) fastens

## TONSILLAR AFFECTIONS.

Every type of tonsillitis met with in temperate climates is observed also in the tropics, although there is no doubt that tonsillar and throat affections are less frequent in warm climates than in cold. Diph-

of Vincent's angina occur, due to Vincent's *Bacillus fusiformis* in association very often with spirochætes. We do not propose giving a description of Vincent's angina which may be found in any text book on general medicine but we would call attention to the possibility of mistaking it for a syphilitic condition. Certain authorities state that Wassermann reaction is positive in Vincent's angina but in our experience this is not so and we can confirm the researches of Taylor and others according to which Wassermann reaction is negative in Vincent's angina except of course when it develops in a syphilitic person. Cases of tonsillitis possibly due to amœbæ and flagellates have also been recorded. We propose saying a few words on certain affections of the tonsils to which little attention has so far been paid—viz. mycotic infections.

Tonsillar nocardiomycosis and lesions of the tonsils due to *Nocardia bovis* and other species of the genus *Nocardia* and Cohn's *reptothrix* have been placed on record but we desire to call attention to the comparative frequency of a granular Nocardiasis of the crypts which may lead to the formation of tonsillar calculi. The affection which is not new, but is little known runs a chronic course and is not painful. The patient often does not come to consult the doctor because of sore throat but because of the unpleasant odour of the breath. On examination the teeth and gums may be quite healthy, but on examining the throat small whitish-



FIG 743.—TONSILLAR AFFECTION CAUSED BY *Hemyspora rugosa* CASTELLANI

yellowish spots will be seen on the tonsils, these spots are in reality the surface portion of granules contained in the crypts and may be extracted with more or less ease. These bodies when squashed have a very offensive odour. Under the microscope they consist of masses of *Nocardia* like organisms at times at other times masses of *Leptothrix*. In certain cases both *Nocardia* fungi and *Leptothrix* are seen and various bacteria and even protozoa such as amoebae and flagellates. The *Nocardia* fungi are very difficult to grow. After several years the masses in the crypts may become calcified and real calculi may be formed which at times are the starting point of some very severe inflammation.

**Varieties of Tonsillonocardiasis**—Clinically the usual form is of the yellow or whitish type. A case observed by one of us was characterized however by the presence of black granules from which *Nocardia* similar or identical to *Nocardia nigra* Castellani 1912 was grown.

**Tonsillar moniliasis**—Moniliasis of the tonsils has been described by Castellani. Three types may be distinguished: acute, the subacute, the chronic. The acute type is important in such cases have often been taken for diphtheria. The tonsils are covered by creamy white patches which at times extend to the soft palate, the pharynx and larynx. There is difficulty in swallowing and the patient may have some fever. Diphtheria is often suspected but the microscopical and cultural examination clearly establish the diagnosis at once. The fungus most commonly found in Ceylon is *Monilia tropicalis* Castellani. Cases of mixed infection of diphtheria and moniliasis have occasionally been seen by us.

In the subacute and chronic types of moniliasis the subjective symptoms are often nil. The diagnosis is based on the microscopical examination. The treatment consists in applications of glycerine of borax and of carbolic acid.

**Tonsillar Oidiomycesis**—The condition is due to fungi of the genus *Oidium* Link. Clinically the affection is very similar to moniliasis but in the case in which *Oidium rotundatum* Castellani was found the patches were yellowish and not white.

**Tonsillar Hemisporomycesis**—In certain cases of tonsillitis in Ceylon Castellani found a fungus which he had previously observed in cases of bronchomycosis. He was doubtful about the classification of the fungus and at first placed it temporarily in the genus *Monilia* naming it *Monilia rugosa* Castellani 1909. Recently Pinhas has placed it in the genus *Hemispora*, the name of the fungus becoming *Hemispora rugosa*.

The case in which the fungus was first observed had been suspected by the house physician to be a case of diphtheria as the patient complained of great pain in swallowing. There was fever, the submaxillary lymphatic glands were enlarged and on examination of the throat several greyish patches were seen on the left tonsil and on the soft palate. At times however the patches are yellowish

but one or two small spots remain often for weeks and even months. As regards treatment painting with a 5 or 10 per cent solution of carbolic acid is found useful.

### GASTRIC DISEASES

All forms of **Dyspepsia** are common but hyperchlorhydria and fermentation are especially common in our experience.

**Ulcers** and **Cancers** of the stomach are met with at times as well as dilatation of the veins at the lower end of the œsophagus leading to severe hæmatemesis. In children **Pyloric stenosis** has been seen several times by us. We have met with one case of diffuse inflammation which resembled phlegmon of the stomach in its gastric symptoms but which was associated with other signs pointing to a more general poisoning of the system.

### EARTH-EATING

**Synonyms** — *Geophagy* *French* Mal d'estomac

### BELYANDO SPEW

**Synonyms** — *Grass sickness* (Western Australia) *Gastric spirochætosia*  
(*English* Black)

**Ætiology** — The cause of the disease is unknown. Black has found a spirochæte in the mucous membrane of the stomach. This spirochæte is of variable size, small and slender.



It is thought that the other members of the family are not

the most common members of the family. The principal symptoms are dysphagia, regurgitation, and vomiting. The suggested are carbolic acid, creosote, and  $\beta$  naphthol. Whatever drug is used it should be given at least half an hour before meals.

**Prophylaxis**—Nothing is known about this.

### ENTALÇAÇÃO

**Synonyms**—Mild engasgo, Dysphagia Tropicale, Tropical Cardiospasm.

**Remarks**—This disease has been known for a long time in certain parts of Brazil having been described by Botelo Langard and by Paranhos. Bouchard has noticed a somewhat similar disease in pheasants which is due to a worm.

**Climatology**—It appears to be localized to some districts in the interior of Brazil.





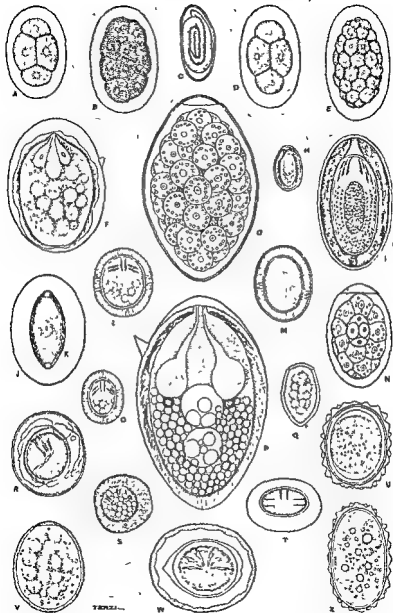


FIG 746—OVA OF INTESTINAL WORMS AS SEEN IN THE FACES

*Albizia anthelmintica* (Bessina) flowers and leaves of *buddleia polistachya* (maltari) the bacchæ of *Mæsa lanceolata* (Saoria) and of *Nyrsine africana* (ssa hto) leaves flowers and fruits of *Celosia erygina* (bellida) efficacious also for ascarides Large doses of melon seeds are also used

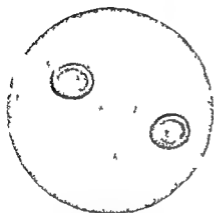


FIG 747—FÆCAL EGGS OF *Tænia solium*

(From a microphotograph by J J Bell)



FIG 748—*Tænia saginata*  
(After Braun)

#### Rare Tæniases

Some of the Tæniases—e.g. those due to *Tænia philippina* *T confusa* *T brevis* *T hominis* and *T africana*—are so rare



FIG 749—*Tænia africana*

(After von Linstov)



FIG 750—*Tænia africana*  
(After von Linstov)

that no further mention is necessary beyond the descriptions which have been given in Chapter XXX p 614

#### The Dibothriocephalases

The Dibothriocephalases are produced by *Dibothriocephalus latus* *D cordatus* and *D parvus* *D latus* (p 604) causes severe anæmia

at times fever associated with serious symptoms but this infection is not common in the tropics. The treatment is the same as for Tænuiasis.

### The Diplogonoporoses

Diplogonoporosis is found in Japan where it is due to *Diplogonoporus granlis* (p. 605) and in Kourmania where it is caused by *D. brauni* (p. 605). The recorded symptoms somewhat resemble those produced by *Diboliorocephalus laevis*.

### Hymenolepsiasis

*Hymenolepis nana* (p. 610) may give rise to severe reflex nervous symptoms but only when present



FIG 73 —*D. bothriocephalus laevis*  
(PROGLOTTIS)  
(After Braun)

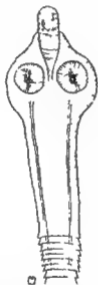


FIG 75 —*D. pylid matricaria*  
(After D'Amare)

in considerable numbers. It has been found in Egypt, Siam, Japan, North and South America, and Europe, and is by no means a rare parasite in man. *H. diminuta* (p. 609) is a common parasite in rats but does not occur so frequently in man, though a number of cases have been recorded in America and Europe. It does not



**Davaineiasis**

<sup>1</sup> *Davainea madagascariensis* (p 611) is normally found in birds but about nine cases have been recorded in children in Madagascar Mauritius Siam and New Guinea

**Intestinal Nematodiasis**

**Definition** — Intestinal nematodiasis is the invasion of the alimentary canal by adult nematode worms

**Strongyloidosis**

**Synonym.** — Intestinal anguillulosis

Strongyloid infection  
p 628)

but induces an intestinal catarrh leading to anæmia and an intermittent diarrhœa when in large numbers The disease is met with all over the tropical world and even in the temperate zone Diagnosis can only be effected by finding the rhabdite embryo (the so called *Anguillula stercoralis*) or the eggs in the fæces in which the latter will only be found when violent diarrhœa is present The treatment is the same as for ankylostomiasis

**Trichuriasis**

**Synonyms** — Whip worm infection Trichocephaliasis

**Nomenclature** — Buttner in 1761 first named the worm *Trichuris* or thread tail for he mistook the posterior end for the anterior Linnæus in 1771 called the worm *Ascaris trichura* but Goeze in 1782 changed the name to *Trichocephalus trichura* because he recognized the error made by Buttner The term *Trichuris trichura* Linnæus must however stand and the term for any disease associated with this worm must be trichuriasis

number of observers who have considered it to be a cause of gastro intestinal and nervous symptoms while Metchnikoff and Guiart in 1901 considered it to be a cause of appendicitis In the same year Girard drew attention to the possible transmission of pathogenic bacteria into the tissues via the wounds produced by the worm In 1902 Schuller ascribed a case of high fever to the action of a heavy infection with the parasite In 1908 Musgrave Clegg and Polk contributed an excellent monograph on the whole subject of trichuriasis together with full accounts of four cases Our own experience is in favour of the worm being occasionally

**Ætiology**—Trichuriasis is caused by *Trichuris trichiura* Linnæus 1771 which as far as is known is really a parasite of man and monkeys while allied forms exist in other animals. The eggs escape with the fæces and require three to six months for the

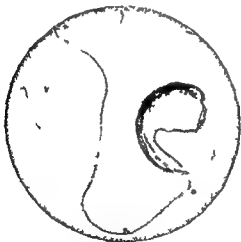


FIG. 753.—*Trichuris trichiura*  
(Microphotograph by J. J. Bell.)

in the tropics where human fecal matter is often allowed to be deposited in vegetable gardens. Musgrave, Clegg, and Polk report finding the ova in washings from fresh vegetables. Domestic animals, insects, flies, dust, etc. are also considered to be possible means of infection. When taken into the alimentary canal it hatches and reaches sexual maturity in sixteen days.

It occurs more commonly among children than adults, women



**Davaineiasis**

*Davainea madagascariensis* (p 611) is normally found in birds but about nine cases have been recorded in children in Madagascar Mauritius Siam and New Guinea

**Intestinal Nematodiasis**

**Definition** —Intestinal nematodiasis is the invasion of the alimentary canal by adult nematode worms

**Strongyloidosis**

**Synonym** Intestinal anguillulosis

Strongyloidosis usually called intestinal anguillulosis is the infection of man with *Strongyloides stercoralis* Bavay 1876 (vide p 628) which produces no symptoms if present in small numbers but induces an intestinal catarrh leading to anæmia and an intermittent diarrhoea when in large numbers The disease is met with all over the tropical world and even in the temperate zone Diagnosis can only be effected by finding the rhabdite embryo (the so called *Anguillula stercoralis*) or the eggs in the fæces in which the latter will only be found when violent diarrhoea is present The treatment is the same as for ankylostomiasis

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**Definition** —Trichuriasis is an infection of the large intestine

number of observers who have considered it to be the cause of gastro intestinal and nervous symptoms while Metchnikoff and Guart in 1901 considered it to be a cause of appendicitis In the same year Girard drew attention to the possible transmission of pathogenic bacteria into the tissues via the wounds produced by the worm In 1902 Schiller ascribed a case of high fever to the action of a heavy infection with the parasite In 1908 Musgrave Clegg and Polk the whole subject of trichi. four cases Our own experie casionally

*Trichuris trichiura* Linnæus  
 usually a parasite of man and  
 other animals. The eggs  
 escape with the fæces and require three to six months for the  
 development of the embryo which can then remain alive for years  
 inside the shell if kept on moist earth. Usually the eggs gain access  
 to man by means of contaminated food especially uncooked vege-  
 tables and to a less extent fruits. This is especially likely to occur



FIG. 753.—*Trichuris trichiura*  
 (Microphotograph by J. J. Bell)

in the tropics where human fecal matter is often allowed to be  
 deposited in vegetable gardens. Musgrave, Clegg and Polk  
 report finding the ova in washings from fresh vegetables. Domestic  
 animals, insects, flies, dust, etc. are also considered to be possible  
 means of infection. When taken into the alimentary canal it  
 hatches and reaches sexual maturity in sixteen days.

It occurs more commonly among children than adults, women  
 than men, and native races than European. But all these factors

stood but the Zoological Committee decided otherwise and we have no choice in the matter and simply carry out the rules for the time being in force. After his discovery the recognition of the disease spread at first slowly but later rapidly. Thus Bilharz in 1853 and Griesinger in 1854 recognized it in Egypt and Wucherer in

by the skin, the lungs, trachea, etc. while more recently Sambon, Fulleborn, and v. Schilling, Torgau, have traced a subsidiary route from the lungs via the blood stream to the alimentary canal. In 1902 Stiles described *Necator americanus* and in the same year Boycott and Haldane found the disease in the mines of Cornwall.

**Climateology**—The disease will be found wherever there is a suitable temperature and moisture for the development of the parasites. It is therefore spread throughout the tropics of America, Africa, and Asia and is also found in Queensland, New Guinea, and Fiji, and also in mines or tunnels in Europe where the conditions of temperature and moisture resemble the tropics.

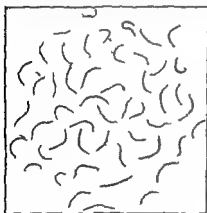


FIG. 754.—*Necator americanus*  
(Natural size)

**Ætiology**—The disease is due to the presence of *Ancylostoma duodenale* and *Necator americanus* in the body. These parasites, as far as is known, live entirely in human beings and are therefore kept alive by patients suffering from the disease and by carriers or

persons infected with so few worms that little or no symptoms are produced. As already noted, the larvae live in earth and infection takes place by two routes—either through the skin or

— " " " " ed by phagocytes at the ovins to the

alimentary canal or inoculated into the blood stream, or on the cephalic glands of the adult worm as it grips the villi of the intestine. But absolute proof is still required of the presence of these toxins, notwithstanding the work of De Giovanni, Loeb, Gabbi, Noc, Alesandrini, and many others. Weinberg's researches on various helminthotoxins must be specially mentioned. The Porto Rico

Commission confirms the suspicion which had long existed as to a relative racial immunity, finding 71 per cent of the cases in Europeans 54 per cent of the cases in mulattoes and 41 per cent of the cases in negroes to vary from medium to very severe though the degree of infection of the three races was in the proportions of 78 per cent, 72 per cent and 76 per cent



FIG 755—CASE OF ANKYLOSTOMIASIS  
(Note the swollen face)

**Pathology**—When the embryos enter the skin in sufficient

It is not known what effect the journey from the skin to the alimentary canal has upon the host, but it is certain that in course of time a marked effect upon the blood and the organs of the body is produced, which often appears out of proportion to the number of worms which can be found in the intestine. Loeb and Smith find that the worm produces a substance which hinders the coagulation of the blood. Some observers—*eg*, Gabbí—have found that the blood of ancylostome patients is more toxic than the blood of healthy people and contains more hæmolytic substances, but this has been denied by Marmi. Padoa and others have observed that the processes of intestinal putrefaction are very marked in ankylostomiasis. In our opinion the anæmia is due to a complex of causes—*viz* the hæmolytic toxins secreted by the worm, actual loss of blood from the bites of the worms, and microbic secondary infections.

sometimes hypertrophy of the left ventricle. In the spleen presents various appearances but generally is shrunken, the pancreas is normal as are the suprarenals, the stomach shows chronic gastritis, the jejunum and ileum are usually contracted, and the mucosa is often dark red in colour and marked by small hæmorrhagic points, which indicate the position of the bites. The ancylostomes may be found in large numbers or may require considerable looking for. Sometimes they are firmly attached to the mucosæ. The kidneys are usually enlarged, pale and fatty.

**Symptomatology.**—The first stage or invasion of the body by the embryos may be marked by dermatitis of various types, papulo-vesicular or pustulo-ulcerative. The dermatitis is generally situated on the soles of the feet and called by the natives of the

second stage is the development of the disease before the definite disease begins. The third stage is when the

dropsical, the pallor being

may be general œdema, with

## SYMPTOMATOLOGY

scrotum or face. When the ascites is marked the patient is unable to lie on his back.

vertigo and floppiness result and in addition there are dyspepsia due to the chronic gastritis and there may be vomiting heartburn and pain over the pit of the stomach. The bowels may be constipated or there may be diarrhoea. *Ascaris* and *Trichuris* together with those of *Ancylostoma* seen in the feces. Occasionally a little blood and mucus may be present and Charcot Leyden crystals may also occur.

**Blood**—There is marked anaemia which has been described by Boycott and Haldane by using Haldane and Lorrain Smith's method of estimating the total volume of the blood to be

index from 0.71 to 0.56 leucocytes from 44,800 to 38,000 polymorphs 43.7 per cent lymphocytes 14.4 per cent mononuclears 5.9 per cent transitional 7.4 per cent eosinophiles 2.1 per cent mast cells 1.5 per cent Ashford King and Gutierrez found erythrocytes from 1,500,000 to 1,000,000 haemoglobin from 8 to 1 per cent leucocytes from 10,000 to 100,000 polymorphonuclears 54.5 per cent lymphocytes 16.3 per cent mononuclears 8.6 per cent eosinophiles 17.1 per cent other 100 per cent

The red cell count is

### oligocythaemia

Boycott has shown that the principal leucocytic change to be seen in the blood before a condition of marked anaemia is oligocythaemia when it is found that there may be high leucocytosis—56,000—with a very high eosinophile figure of 56.2 to 62.2 per cent while the haemoglobin was from 9.8 to 5.1 per cent. When

to be explicable by the hydræmia producing the anæmia. Boycott however considers that this is not so for he points out that leucocytosis can occur with marked anæmia and that there is always a tendency on the part of the blood to restore its average volume and composition when altered from the normal and comparing the normal leucocytic count of the hydræmia of chlorosis says that if there was no other factor the leucocytic count of ankylostomiasis would not be affected by the hydræmia.

He thinks that the true explanation is probably exhaustion of the bone marrow produced by the anæmia and that it is partially due to a failure on the part of the individual to react to the stimulus to produce the eosinophile leucocytes. In any case the leucocytic reaction does not bear any relationship to the anæmia. According to Boycott and Haldane if the eosinophiles are deducted from the total number of the leucocytes and the percentage of the remainder then calculated it will be found to be nearly normal. The eosinophilia has been found to remain after the ova have quite disappeared from the fæces. Any inflammatory complication which leads to a polymorphonuclear increase may hide the true eosinophilia. The leucocytes are normal in structure a few neutrophile but no eosinophile myelocytes are to be seen. Wernberg and Mello have worms

at that  
of the

eosinophiles and at the same time preventing a proper oxidation of hæmoglobin and finally producing the hydræmia. We must state however that occasionally we have come across severe cases showing no eosinophilia. Low has demonstrated that eosinophilia is generally well marked in children while it may be absent in adults and suggests that the eosinophiles which are tissue cells at first come into the blood in response to some stimulus set up by the infection but gradually disappear as this stimulus weakens with the prolongation of the infection. As a result of the diminution of the production of hæmoglobin the iron in the liver is diminished.

Patients often complain of palpitation or difficulty in breathing. The lungs will be found normal but the heart may be displaced downwards and to the left and be feeble with a hæmic bruit at the base. The vessels of the neck may be seen to pulsate markedly. The pulse is quick and may be weak, thready, dicrotic and intermittent. The liver is very often enlarged especially in children.

*Fever*—Fever in ankylostomiasis was described years ago by

three types of ankylostomiasis fever may be met with—

1. The low intermittent type which is the commonest and in which the temperature seldom rises above 100° F.

2 An irregular type, at times intermittent, and at times sub-continuous

3 An undulating type This is very rarely observed.

Great care should be taken in making the diagnosis of ankylostomiasis fever to exclude other conditions—eg, malaria, Malta fever, kala azar, trypanosomiasis etc. There is much diversity of opinion on the origin of this fever. In our experience the fever

quite appropriate. In several cases the fever continues long after the patient has got rid of the ancylostomes by adequate treatment.

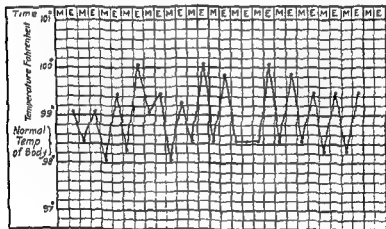


FIG. 756.—TEMPERATURE CHART OF A CASE OF ANKYLOSTOMIASIS FEVER.  
(From a case in the Clinic for Tropical Diseases, Colombo.)

**Urine**—The urine is copious, pale and often alkaline, with a specific gravity varying from 1010 to 1015. Albumen is rarely seen, but there is an increase of indigo-blue and urobilin and there is albumosuria at times. The excretion of nitrogen is said to be much increased. Lussana believes that there are toxins in the



Mental and physical hebetude are marked and other nervous symptoms may be observed. Children do not develop properly and sterility with delayed menstruation has been noted.

When the anemia becomes profound death may ensue from cardiac failure but any intercurrent affection hastens the end in a case of ankylostomiasis.

**Complications**—Any intercurrent disease is a serious complication as also is pregnancy.

**Diagnosis**—The definite diagnosis depends upon finding the ova in the feces.

Anæmia in tropical countries especially if associated with dropsy should at once lead the practitioner to suspect ankylostomiasis. It must be remembered however that it is by no means easy to find the ova in the feces at the first examination and that sometimes even in the worst cases they require to be looked for on several days. Occasionally it is useful to give an aperient to make the eggs appear in the stools.

Several methods have been used but the most reliable is based on the following: A small quantity of feces is placed on a filter paper and after the deposit has been made on the filter paper the filter is placed on a glass plate and the feces is allowed to dry. The filter is then placed in a test tube and the tube is shaken for several minutes. The deposit is then examined under a microscope.

**Bass's method** is to dilute the feces with ten or more times their bulk of water and centrifuge the mixture for three or four minutes. The supernatant fluid is then examined under a microscope.

**Prognosis**—There is no doubt that ankylostomiasis is one of the great factors in producing the death rate of a tropical native. It is often not diagnosed and the patient is treated for malaria, general dropsy and malarial cachexia. Ashford, King, has reported a case in which the patient was cured by ankylostomiasis. We are not at all sure that the disease is

frequently entered in death certificates as anemia, general dropsy and malarial cachexia.

**Treatment**—The aim of the treatment is to kill and remove the parasites and this can be effected by thymol, eucalyptus oil, eucalyptol, beta naphthol or male fern. A case must not be considered cured until the feces show no ova on repeated examination after two or three weeks.

In all cases the patient should be carefully examined as to the condition of all his organs before treatment is begun and should be placed on low or liquid diet for a day or so and while being

treated should be kept in bed and care taken that the bowels have been well opened

*Thymol* introduced by Bozzolo should be given in cachets or as an emulsion. Generally 15 to 30 grains are given and two hours later another 15 to 30 grains followed in some cases by a third dose of 15 to 30 grains after another two hours. If the bowels do not act within four hours of the last dose a saline aperient should be given. The treatment may be repeated on the following day. Another method is to give 10 grains in cachets at night until the desired result is attained. Yet another method recommended by the International Health Board is to mix it with an equal quantity of bicarbonate of soda as this addition is believed to aid the cure and prevent unpleasant symptoms.

*Thymol* is a very poisonous drug in large doses causing first irritation of the cerebral centres with excitement and vertigo

collapse

*Thymol* however is very insoluble in water—only 1 in 1500 of cold water—but it is easily soluble in alcohol ether chloroform glycerine and turpentine hence no alcoholic stimulant whatever must be given to a patient who is to take or has taken *thymol* and not merely must care be taken not to order stimulants but the nurses must be warned of the danger of giving them. *Thymol* is also soluble in oil. Hence no purgative of castor oil should be ordered after its administration. *Thymol* certainly should never be given if there is marked visceral disease nor do we think that it should be given in very profound anemia—i.e. when the number of red cells is below 150000 per cubic millimetre. It is however very satisfactory in its lethal action on the worms but the treatment must be repeated in many cases in a week and again repeated if ova are seen in the feces. Sandwith recommends a hypodermic injection of strychnine before *thymol* is administered.

*Eucalyptus Oil and Chloroform*—A much less dangerous treatment is by oil of eucalyptus and chloroform which may be preceded by a saline purgative given a few hours earlier.

The usual formula is —

Olei eucalypti  
Chloroform's  
Olei ricini

℞xxx  
℞xl  
ʒx

One half to 1 —

Messrs Freudenberg and Company of Colombo use a simple incinerator and they have kindly supplied us with the following details of their process which is carried out at night —

Average number of buckets of faecal matter burnt per night 20

The urine can of course be separated from the faeces by using a Donaldson separator latrine. The urine can then be disposed of by burial and the faeces burnt.

There is no doubt that some such method would have excellent results in dealing with the problem in small communities and estates if carried out properly.

Badly infected lands might be treated with lime. It has been recommended that coolies should protect their feet by first dipping them in a bucket of tar and then in one with sand.

Patients attend at a central or one of the outlying depots or dispensaries where their stools are examined and they are given medicines and a card with the following instructions —

- 3 Take the other half at 8 a.m. the same morning
  - 4 Take the other purgative at 10 a.m.
  - 5 You should neither drink wine nor any alcoholic liquor during the time you are taking these medicines.
  - 6 Come for more medicines until the physician says you are cured.
- Do not defæcate on the surface of the

Salt has also been recommended by Perrot and others but as a solution of at least 2 per cent is necessary to kill the larvæ this method is too expensive.

## ASCARIASIS

### SUMMARY OF PREVENTIVE MEASURES

#### Educational —

Instruction of rich and poor with regard to the symptoms and treatment of infection of the small intestine.

- 1 Search for and treatment of carriers
- 2 Search for cases of the skin eruption and treatment of the same
- 3 Search for cases of anemia and treatment of same
- 4 Provision of sanitary conveniences kept in good condition also associated with a good system of conservancy

### Ascariasis

**Definition** — Ascariasis is infection with the round worm, *Ascaris lumbricoides*.  
Linnaeus 1759

Zeder 1800

#### Symptoms

those of intestinal obstruction being present. In some cases the reflex white spots may be seen.

however

toxic signs

of the worm may develop but the great danger is from wandering of the parasites. If they wander up the bile duct into the liver they may cause abscesses in the liver. We found as many as eleven worms in the bile-duct with a number in the liver three of which lay in abscess cavities may also enter the duct of Wirsung and cause slight inflammation of the pancreas or go into the appendix and cause appendicitis. Further they may pierce the bowel in cases of ulcerative condition of the bowels, and enter the peritoneum or the bladder or enter the nose or the ear by the Eustachian tube — or wander all over the body.

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seen in cases which on post mortem examination have shown no lesions of typhoid but enormous numbers of ascariasis.

**Diagnosis** — The diagnosis will depend upon the discovery of eggs in the feces.

**Treatment** — The best treatment is santonin with some purgative. Usually santonin is mixed with an equal quantity of ...

appear in the fæces. It is as well to remember that *santonin* may cause blue or yellow vision.

Oil of chenopodium may be given in gelatine capsules or in castor oil (for dosage see p. 1770). In China *Quisqualis indica* is at times used. 2 drachms of the powder.

### Oxyuriasis

**Definition**—Oxyuriasis is infection with *Oxyuris vermicularis* Linnæus 1767 (see p. 857) and is common all over the world.

**Symptomatology**—The symptoms are irritation in the region of the anus with sometimes a distinct entero colitis and sometimes slight fever.

In the nose  
the motions a

forms require internal treatment with *santonin* and calomel as described under *Ascariasis*. Flynn recommends sulphur (gr. iii)

and naphthahn suppositories may be used.

### Intestinal Polyparasitism

**Definition**—Intestinal polyparasitism is the invasion of the alimentary canal by more than one species of parasite.

**Remarks**—We have since 1903 investigated the question as to which parasites are commonly present in natives of tropical Africa and Ceylon and find that it is the rule rather than the exception for their intestines to harbour more than one species of parasite.

Statistical information with regard to the prevalence of the various forms in different tropical regions is still wanting but some valuable observations have been made especially in the Philippine Islands by Garrison and in South Africa by Miss Porter. The relative prevalence of the various intestinal parasites in the Philippine

The prevalence of the various parasites were as follows—

	Per Cent.
Trichuris	59.0
Ascylostoma and Necator	52.0
Ascaris	26.0
Amœbæ	23.0
Flagellates and Ciliates	21.0
Strongyloides	3.0
Oxyuris	0.8
Tænia	0.7
Schistosoma japonicum	0.6
Paragonimus	0.4
Opisthorchis	0.3

Besides these however, there were a number of undetermined forms. — even in different countries — 1 per cent India (Fearnside) 1 per cent Central Africa the *Ancylostoma* and *Necator* is given in India (Wellman) 65 per cent (Dobson) 57.58 per cent (Fearnside) 65.83 per cent *Ascaris* infection in West Africa (Wellman) is 50.97 per cent Garrison considers his figure low 15.37 per cent

**Ætiology** — *Ascaris lumbricoides* is in our experience by far the not uncommon and quadruple infections of the three associated no means *Trichostrongylus axei* *Oxyuris equi* is are not so frequently met with in Ceylon India and Equatorial Africa while they are extremely common in Abyssinia

In China and other countries *Trematode* infections must also be considered and in the West Indies and Africa infection with *Schistosoma mansoni*

**Symptomatology** — The symptoms presented by the patients may be nil if the parasites are few in number and will in any case depend mostly upon the action of that species which is known to be the more pathogenic or which is most abundant but it may be very difficult or impossible to separate the symptoms caused by one parasite from those due to another. Cases may show signs of fever, anæmia, diarrhoea and even dysenteric symptoms may appear if the infection is heavy

**Treatment** — The treatment must commence with that laid down for the parasite which is the more important from a pathogenic point of view — in the case of a double infection with *Ancylostoma* and *Ascaris* the ankylostomiasis must be treated first and then the ascariasis.

### Rare Infections

**Gordiasis and Acanthocephaliasis** (see pp 6 8 and 679)

Infections with species of the Gordiacea and Acanthocephala are rare Treatment would be on the same lines as for ascariasis







## CHAPTER LXXVI

# SPRUE AND OTHER DIARRHŒAS

Sprue—Pseudo Sprue—Hill diarrhœa—Low country morning diarrhœa—  
Flagellate diarrhœa—Famine diarrhœa—Cœliac disease—References

### SPRUE.

**Synonyms.**—Ceylon sore mouth, Aphthoides Chronica, Tropical aphthæ, Impetigo Præmarum Viarum Diarrhœa Alba Psilosis Lingue et Mucosæ Intestini Phthisis Abdominalis Blastomycosis Intestinalis Endemic diarrhœa Cochîn China diarrhœa

**Definition.**—Sprue is a chronic catarrhal inflammation of the alimentary canal, of unknown cause, characterized by a peculiar ulcerative condition of the tongue and mouth, and by the passage of large, pale, frothy motions, the symptoms waxing and waning periodically

**History.**—According to Hiatt, sprue was first mentioned in the writings of John Bicknell, in America, in 1737. Hillary, of Barbados, in 1766, in a most remarkably able manner, describes the disease for the first time under the name 'aphthoides chronica'

mention symptoms indicating that they were acquainted with a disease of this nature, while Elliott of Ceylon gave a very good account of the malady, which he called 'phthisis abdominalis'

At a later period, physicians noted the disease in Cochîn China, believing that it was probably a new disease

In 1880 Manson was the first after Hillary to clearly define the disease, which he called 'sprue', and in the same year, and independently, Van der Burg described it under the term 'Indische spruw' in Batavia. In the next year (1881) Sir Joseph Fayrer

occur in Europe

Ætiology.—The etiology of sprue has not yet been elucidated, but of the many etiological theories brought forward, the one which at the present time receives more acceptance is the monilia or oidium theory, also known as *Kohlbrugge's theory*. Kohlbrugge, in 1901, found in cases of sprue in Java a fungus which he identified with *Monilia albicans* Robin, at that time better known under the name of *Oidium albicans*. He made a very complete histological study of one of his cases which ended fatally, and

In 1905 and 1912 cases of sprue with presence of monilia fungi were placed on record by Castellani who, in 1912, described several species, *Monilia intestinalis*, *M. enterica*, etc. This author's opinion was that such fungi were the cause of some of the symptoms of

large doses might decrease the acidity of the intestinal contents, and in this way check the growth of fungi, which, as is well known, grow better on acid than on alkaline media. In 1913 Castellani and Low described a new monilia found in a case of sprue, *M. deco-*

the primary cause, the acids resting publications, supported the cause of the malady was

ible papers, has supported the



were of the viridans type as found also in normal mouths. Complement fixation tests carried out by Nicholls would seem to support the streptococcal theory. Nicholls believes the etiology to be in reality twofold there being an infection factor and a dietary factor.

Attention must be called to certain cases of pseudo-scurvy,

found in cases of sprue in which spirochetes, flagellates, etc., but none have been demonstrated to be the cause of the malady.

**The Climatic Theory**—This does not need to be discussed though a hot damp climate is an important predisposing cause.

**The Food Theory**—This also does not need to be discussed though spicy foods and alcohol may be predisposing causes and dietary errors as emphasized by Nicholls may lower the resistance of the alimentary mucosa to germ infection.

**The Deficiency Theory**—This theory has been ably brought forward by Cantlie who noticed in certain cases signs of scurvy.

**Syndrome Theory**—Finally, the theory must be mentioned

medical man of long tropical experience. The disease presents

direction and the absorption of the products must be interfered with, while at the same time the absorption of poisons from the

same theory, though he does not consider that *M. albicans* is the cause of the malady. He calls the monilia observed in his cases "fecal," but according to the laws of nomenclature the correct

149

- 1 *Monilia albicans* Robin
- 2 *Monilia decolorans* Castellani and Low.
- 3 *Monilia intestinalis* Castellani
- 4 *Monilia fecalis* Castellani
- 5 *Monilia insolita* Castellani
- 6 *Monilia tropicalis* Castellani
- 7 *Monilia enterica* Castellani (probable synonym  
*Monilia psilosis* Ashford, *Parasaccharomyces ashfordi* Anderson)

For description of these fungi see p 1079

Species of the Genus *Oidium* found in Sprue.—The principal species of the genus *Oidium sensu stricto* so far found in sprue are—

- Oidium rotundatum* Castellani
- Oidium asteroides* Castellani

For description of these fungi see p 1093

Remarks.—Kohlbrugge's theory is the one finding most support at the present time and according to various authors, agglutination and complement fixation tests are supporting it, and certain observers have claimed to have succeeded in reproducing the malady in the lower animals by injection of intestinal monilias. We believe that if a group of monilias and not one only, will be found to be capable of producing the affection this in analogy to what one sees in bacillary dysentery and in affections due to the higher fungi such as ringworm.

It must be also in stools which are not *Monilia Sacca*

oted that fungi of normal individuals connected with *Saccharomyces Crypticus*

— he found nos era

this Theory.—Some authorities consider *Strongyloides* to be the cause of the disease, but in our experience the thing to do with the malady, being found in all sorts of conditions

1 Theory.—Numerous different cocci, bacilli, etc., are named as the causative agents of sprue, but so far demonstrated to be the primary cause of the malady. Nichol and Rogers have suggested that the disease may be a streptococcal infection, both authors having obtained good results with bacterial vaccines. The streptococci found by Nichol

The Helminth *Strongyloides* to which has not been described in the pathologic literature. The Bacteria have been described by Rogers and Nichol as *Streptococcus* by using

were of the viridans type, as found also in normal mouths. Complement fixation tests carried out by Nicholls would seem to support the streptococcal theory. Nicholls believes the etiology to be in reality twofold there being an infection factor and a

discussed causes and resistance

of scurvy mentioned

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could appear as though the

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the glands and villi being affected

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with white patches

The lesson we have drawn from the above is to get a sample analyzed from time to time. This analysis costs but little and the possibility of its being carried out at any time puts a certain amount of restraint upon would be adulterators.

In placing a patient upon a milk diet the composition of the milk should be carefully considered especially as regards the fat, for, as Harley and Goodbody have shown, no less than 47 per cent of the milk fat is passed out in the feces.

Milk with high percentages of fat should, therefore, be diluted with whey, when the amount of nitrogen will be kept up, while the percentage of fat is diminished. Whey is easily made in the tropics by means of the juice of limes.

Preferably the milk should not be boiled or sterilized but boiling, apparently, does not interfere with its beneficial properties, and, therefore, if desired or thought necessary—owing to the risk of typhoid—there is no harm in so doing. In cold weather it should be warmed before being taken. It can be aerated in a

or it can be mixed with Vichy water. Finally, for an adult,

gradually to increase the amount. Every medical man sooner or later adopts his own method of carrying this out and we will therefore, only give general directions. If the case is very severe with vomiting and much diarrhœa, it is as well to begin with whey only, which the patient should sip slowly and practically *ad libitum*—about 7 to 8 pints per diem. As soon as the urgent symptoms are relieved milk must be added to the diet as whey alone is starvation.

If the case is of moderate severity milk can be begun at once, 3 pints per diem being given in the more severe and 4 pints in the less severe cases—60 to 80 ounces—which should be divided into not less than ten meals at regular intervals during the day.

On no in gulps at twenty

If the symptoms improve it is necessary to increase the milk to 120 to 140 ounces—to increase the number of meals. Twelve meals in the twenty four hours are not unusual to arrange.

If the symptoms do not improve, the milk must be reduced gradually, or whey must be tried, but as soon as the urgent symptoms of

Starvation diets are advised and hence while they are being

ng  
ceases the mouth troubles

The feces will at first be  
continues stercobilin will

appear as evinced by the brown colour

This change in the colour

the patient there will be much  
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must be relieved by enemata  
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alkalinizing the milk with bi-

or the citrate of soda 2 grains to

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but great

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Hanbury's Mellin's Albany  
togen or Plasmon Then fine  
biscuit and later potatoes

a diet of eggs toast dilute China tea soups white meats

most vegetables

Alcoholic

afterwards

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milk may be

exceedingly

mentioned

concerning one of which we have our own experience,  
concerning the other none at all First of all there can be no  
reasonable doubt as to the advantages of liver soup—the soup  
prepared from calves or sheep's livers—in the milder cases of sprue  
or in the return to ordinary food of a severe case The liver treat-  
ment is really an old native remedy in Ceylon We do not profess



by some, but of these we have no experience

(b) *Milk and Fruit Diet*—A milk and fruit diet has been found to be even better than a pure milk diet in many cases. The milk

crushed, and eaten with sugar and cream. In lieu of strawberries,

the strawberries or apples, from  $\frac{1}{2}$  pound to  $1\frac{1}{2}$  pounds per diem

may be boiled in water and then cut open and shredded by means of a fork into warm milk, in which it is pounded with castor sugar, and finally strained through a fine strainer to remove all debris. This should be used three times a day. Preserved fruit is said to be useful, if fresh fruit cannot be obtained. Acid fruits, such as pineapples and sour sops, should be avoided, and, personally, we do not advise the use of mangoes.

(c) *Fruit Diet*—This was first advocated by Van der Burg and usually

if successful

in  $1\frac{1}{2}$  drachm doses every morning for the first three days and santoun in  $\frac{3}{4}$  grain doses morning and evening for three days Strawbernes to the

6 MEDICAL TREATMENT—It may be said at once that astringents as a rule are dangerous and that antiseptics are not useful

REINHOLD TO ELLER WEEKLY

Castellani has obtained in some cases a remarkable improvement by giving massive doses of bicarbonate of soda

The mouth may be treated with any appropriate mouth wash—*e.g.* glyco thymoline, glycerine and borax diluted liquor aluminis, etc The teeth require attention and if there is any pyorrhoea,

\* Gus requires morphia Janowski recommends 5 to 10 drops of a solution of 1 in 1,000 adrenalin, which, he says, gives prompt and

checked by a dose of liquor opii sedativus or lead and opium pills. Dysenteric symptoms must be treated as described under Dysentery

7. CHANGE OF CLIMATE—It is obvious that if possible the patient should be transferred from the tropics to the temperate zone, but only if he is strong enough to travel. There is no advantage in putting him on board a ship in such a condition that he will probably die when changing from the warm to cooler weather. Personally, we are not in favour of a patient being sent from the low country to the hills. If he is able to travel, let him go to the temperate zone.

VACCINE—Various bacteria from the mouth lesions of patients with streptococci isolated from the mouth lesions. Monilia vaccines have been used by Ashford Michel Taylor and others, who claim satisfactory results.

Prophylaxis.—Nothing of any practical value can be said under this heading.

### PSEUDO-SPRUE.

One of us has called attention to some cases presenting clinical symptoms closely allied to sprue and due to bacilli of the Flexner group. Such cases have no dysenteric symptoms but present the white frothy motions, the sore tongue and the anæmia as found in typical sprue but in contrast to this disease they may recover without any change from the tropics and a vaccine treatment is very useful.

Motility	Lactosa Milk	Lactose	Saccharose	Dulcose	Mannite	Glucose	Maltose	Dextrin	Raffinose	Arabinose	Adonite	Inulin	Sorbito	Galactose	Lactulose
o	A	o	o	o	A	A	A	A	A	A	o	o	o	A	A
Inosite	Saltum	Amalgam	Isodulcose	Erythrite	Glycerine	Indol	Voges Prosk	Redm Nitrites	Neutral Red	Gram	Gelatine	Serum	Broth	Pept Water	
o	■	o	A	o	As*	+s	o	o	o	o	o	o	GT	GT	

Abbreviations—A acid GT general turbidity ■ slight ■ negative result—viz neither acid nor gas in sugar media non motile non liquefaction of gelatin or serum as the case may be

\* Certain strains are distinctly acid on ninth day

## HILL DIARRHOEA.

believed the monsoon to be a potent factor, associated, probably, with a diminished barometric pressure. We have seen cases in Ceylon occurring at a much less elevation—for example, at about 3 000 to 4 000 feet.

More recently Duncan has put forward the view that mucus in

to invite attention to what has already been written under the

lymph and fibrous tissue in chronic cases. On the surface of the mucous membrane of the small and large bowels there is a thick layer of mucus but no ulceration.

soon after the

in in the early  
rothy, greyish

The patient goes about his work, but in the early hours of the next morning the symptoms are repeated, and he will complain that his stomach feels blown out, and that he can hear gurgles and this goes on morning after morning.

If now the patient leaves the hills and comes down to the plains

to consult a doctor he is astonished to find that he is quite well and perhaps goes back to the hills without having obtained the medical advice which he desired. A relapse takes place for which he does not as a rule seek advice as he considers it a trivial complaint until later he begins to feel dyspeptic disinclined for his food or work and now he will seek treatment notwithstanding the fact that he feels better on returning to the plains. The disease may become chronic and rarely may lead to a fatal result.

**Sequela**—It is said that neglected hill diarrhœa may develop into sprue.

**Diagnosis**—The history of the case and the absence of mouth symptoms are sufficient to enable the diagnosis as a rule to be made from sprue.

**Prognosis**—The prognosis is good as recovery is generally quick under suitable treatment but in certain cases it is found necessary to abandon residence at high elevations.

**Treatment**—The treatment is simple and effective. It consists in rest in bed warm clothing and  $\frac{1}{2}$  to 1 drachm of liquor hydrargyri perchloridi given fifteen minutes before each meal and 12 to 15 grains of pepsin, ingluvin or lactopeptin two hours after the meal. The diet is to be milk which may be diluted as advised in the treatment of sprue.

**Prophylaxis**—Persons liable to the disease should avoid the hills especially in the monsoon season and if compelled to go to high altitudes should do so by easy stages.

### LOW-COUNTRY MORNING DIARRHŒA

<p>Common in Ceylon and the plains</p> <p>3 to 4 a.m. with is no abdominal</p>	<p>Common in Ceylon and the plains</p> <p>3 to 4 a.m. with is no abdominal</p>
--	--

yellowish or brownish and do not contain blood or mucus. The condition lasts as a rule for months but usually ceases on a change of climate.

**Treatment**—A dose of tannalbin (gr. xv. xxx) or bismuth subnitrate (gr. xv. xxx) may be given at bedtime as a palliative.

### FLAGELLATE DIARRHŒA



FIG. 759.—*Cercomonas longicauda* DAVINE

(After Wenyon and O'Connor from the publications of the Wellcome Bureau of Scientific Research)

Illustration of Cercomonas longicauda

but if one of these organisms is present in very large numbers in a case if bacteriological research fails to demonstrate any pathogenic bacteria and no other cause can be found it may be provisionally admitted that they are causal. If the causal organism is killed off and the diarrhoea ceases *pari passu* with this process and does not return and the flagellate is either absent or only present in small numbers the first assumption receives support but beyond this we can not at present go.

The difficulty is that the numbers of the parasites wax and wane in the carrier without producing symptoms but when present in large numbers they are generally associated with diarrhoea. Infection may be by the cysts passing into the alimentary canal of flies and so to human food but perhaps it may take place more directly at times. We have never seen them cause true dysenteric symptoms.

From certain experiments carried out by Miss Porter it would seem that cockroaches may play a rôle as transmitters of flagellate diarrhoeas of man. This observer succeeded in transmitting *Cardia Trichomonas* and *Chilomastix* of human origin to clean white rats by allowing their food to be contaminated with the excrement of cockroaches (*Periplaneta americana* and *P. orientalis*) which had fed on infected stools.



FIG. 759A.—Cyst of *Cardia intestinalis* IN FRESH CONDITION IN HUMAN FECES

(Photomicrograph  $\times 600$  diameters)

Another method of treatment is to administer calomel at night, a saline

infections



may be killed after being caught by means of a daisy killer or any other smoke apparatus

All fly breeding places should be destroyed by the removal of the dirt and the digging up and disinfection of the ground

#### FAMINE DIARRHŒA.

**Historical and Geographical.**—This condition has been observed in India during periods of famine and recently by us in

has been found

**Symptomatology.**—The patient is extremely weak and terribly wasted, though

cholera germs

**Prognosis**—This is serious many cases terminating fatally

**Treatment**—This is very unsatisfactory Astringents such as bismuth subnitrate etc even when given in massive doses may not stop the diarrhœa at times they may check it but the patient continues to become weaker and weaker and often dies

## CŒLIAC DISEASE

It in contrast to what one sees in true sprue is badly tolerated and should never be ordered As regards drugs Stille's mixture is often found useful

Oil ricini	℥v
Salol	gr 1½
Spir chlorof	℥i
Muc acaciæ	℥xv
Aq anethi	ad ℥i

Ter die

Occasionally silver nitrate is valuable when the diarrhœa is very marked and Still recommends it to be given thus —

Arg nitratu	gr ½
Glycerin	℥v
Aq dest	ad ℥i

Ter die

Bismuth preparations are practically useless but tanalbin has at times a favourable action

## REFERENCES

### Sprue

The current literature may be found in the Bulletin for Tropical Diseases





## CHAPTER LXXVII THE CHOLERAS

*Cholera*—Synonyms—Definition—History Geography and Epidemiology—  
Aiology—Pathology—Symptomatology—Diagnosis—Prognosis—  
Treatment—Prophylaxis—Paracholera—Pseudocholera—References

### CHOLERA

**Synonyms**—Cholera Asiatica, *Hindustani* Haisa, *Tamil* Enerum Vandee,  
*Chinese* Ho loua, *Arabic* Duba.

**Definition**—Cholera is an acute specific endemic or epidemic disease caused by *Vibrio cholerae* Koch, 1853 and characterized by violent purging vomiting muscular cramps suppression of urine, and collapse.

**Remarks**—There is little doubt that in the past the term cholera—in analogy to other diseases—has been used to cover a group of clinically similar affections caused by closely allied germs. See remarks on paracholera p 1819.

**History, Geography, and Epidemiology**—Cholera appears to have been known in India from the most ancient times for Charaka and Susruta describe symptoms which most probably refer to this disease. The name is of Greek origin being perhaps derived from *cholera* a spout which may have been applied with the idea that the violent purging resembled the water rushing out of a spout. Apart from the two authors mentioned above the earliest record of the disease is found in 1438 when Ahmed Shah's army is said to have been decimated by it. After this date there are several references for it is mentioned by Vasco da Gama in 1498 an account is given of an outbreak in Goa in 1543 another in Pondicherry in 1766 and another in Calcutta in 1781-82 the last of which appears to have spread to Madras Ceylon and Burma in 1782-83. Out-

With regard to Europe cholera was recorded at Nismes in 1564 but it and the sporadic outbursts in the seventeenth century may not have been true cholera. It also appears to have been endemic in Java as far back as 1629 and occurs yearly in Southern China and the Philippines Islands.

In 1817 began an epidemic which may have originated in Calcutta or in Jessore and which lasted till 1823. During this time

it spread to the west coast of India Arabia Ceylon Burma Malacca Penang Singapore and Manilla reaching Mauritius in 1819 and China in 1820 This is certainly the first extensive epidemic ever recorded outside India In 1826 the first pandemic which lasted till 1837 and spread into Europe Africa and America began in India through which it spread slowly and then passed to Europe and Africa by three routes the first and earliest via Kabul Bokhara

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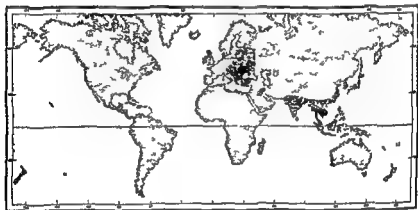


FIG 761 — DISTRIBUTION OF CHOLERA IN 1915

it spread reaching Northern Burma in 1842 It now proceeded north of the Himalayas reaching Yarkand from whence it passed

so to Europe and America in 1848 In this year it was very pre

valent in India, spreading from Bombay to Aral in 1827 and to Turkey in 1853; Russia in 1852, and so on and affected the troops en route.

The fourth extensive epidemic began in 1863, and spread to Europe by the two usual routes—viz, via Persia and Arabia—and

issed from  
epidemic

in India to the great bathing festival at Haragota, on the Ganges, which, being only held once in thirty years came as a surprise to the officials, who were not prepared for it. Cholera broke out among the pilgrims on February 8, 1891, and spread rapidly, reaching Europe in 1892.

The seventh epidemic began in 1900, and may be said to be continuing at the present time. It started in 1900 in India after a severe famine, and spread extensively through that country, and then began to travel reaching Japan in 1901, Mecca and Jeddah, E. Philippines in 1902, Persia, Russia and the Philippine Islands in 1905, Russia (few cases) Burma, Siam, Singapore, Japan, China, and the Philippine Islands in 1906, Ceylon, Peshawar, Singapore, Philippines, Japan, Korea, Manchuria, China, Russia, Turkey and Persia in 1907, China, Russia, Mecca, and Medina in 1908, and Russia in the beginning of 1909. In 1910 it was present in Russia and Italy. In 1911 it was recognized in Turkey, Roumania, Hungary, Austria, Italy, and Russia, and in 1912 in Turkey, Italy and Russia.

In 1914 the Dutch East Indies were attacked, and during the war the Austrian Army and civil population were attacked, especially in Galicia as well as Bulgaria and Greece, while Turkey

lands to which the disease is less likely to be brought, or where

the same year treated six cases similarly without a death. In 1884 J W Fry stated that the only an enema of diluted Condy's favourably of the use of the cholera. In 1913 Castellani d

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have been a fruitful source ay be we do not definitely know, but it would appear that such factors as mental worry *underfeeding* and any *slight disorder* but specially any derangement of the alimentary canal producing diarrhoea are predisposing causes. With regard to these causes it may be mentioned that unripe fruit and especially melons are regarded with suspicion in times of epidemics probably because they cause diarrhoea, and thus predispose to the disease.

It may be that the vibrio merely lives in the lumen of the bowel in the cases in which it causes no symptoms and only gains access to the epithelial cells and mucosa in cases of lowered vitality, and perhaps is only toxic in this situation but if this is so it still remains to be proved.

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on

that manual labour to perform that they are exhausted at night. Therefore if a carrier is introduced into a gaol the disease may easily start apparently *de novo* among incarcerated inmates.

The disease is communicable directly from man to man by contamination. As an example from our own experience a medical man examines a patient suffering from cholera and then proceeds to have his lunch, with the result that both patient and doctor were dead within twenty four hours of cholera. Again there is the chance of infection from dead bodies in performing autopsies but

time (163 days), hence the great danger of faecally contaminated fields, rubbish heaps, gardens, etc, for if there is a poor sanitary

## ETIOLOGY

tem in the place and if faecal pollution of the drinking water is possible the disease may easily become epidemic for it has been shown that the vibrios can not merely live but multiply in water though the conditions under which they do this are not perfectly understood.

Two classical instances are usually quoted as evidence of the spread of the disease by water. The first of these is the infection in 1854 of a lady and her servant in Hampstead where there was no cholera by drinking the water of the Broad Street well which was infected the water being carried all the way from Broad Street to Hampstead because the lady in question had a special liking for it. The second is the infection of Hamburg in 1892 from the waters of the Elbe in which cholera vibrios were found in the river water and that of the hydrants.

As water is a method of infection it is quite easy to understand that milk is specially dangerous for it is often diluted with water and moreover forms an excellent medium in which the germs can grow. Thus Haffkine and Simpson found that an outbreak of cholera in the Gaya Gaol was due to the contamination of the milk, from which they obtained the vibrio.

**The Carrier**—Of great importance in the dissemination of the milk are the so called carriers—viz persons who though themselves in good health still harbour the germ in their intestines or individuals who continue to harbour the germ for months and years after an attack is over.

Greig has demonstrated that carriers can show an increased titre for the agglutination of the vibrio. His researches also show that the vibrio can live for long in the gall bladder of animals and he has also demonstrated it in the human gall bladder.

The fact that the germs can live for a long time in faecal matter regard to flies the germs have been found not merely on the exterior of the body but also in the alimentary canal in which they are believed to multiply. The habits of flies make them therefore an important possible means of dissemination of the disease.

Berber in 1914 brought forward experimental evidence showing that the germ could live for a time in the alimentary canals of *Periplaneta americana* (the cockroach) and *Monomorium lalinod* Myr (the red ant).

Faecal matter may also pollute green vegetables for in the Fa vegetable gardens are often contaminated with human faecal matter. The most dangerous vegetables are those which are eaten raw such as lettuce, watercress and tomatoes. While however the above methods explain many points in the epidemiology of cholera they do not afford a full explanation of the spread of the disease. As we have already said the principal endemic centre is Lower Bengal whence it can spread through India and indeed over the greater part of the world by human agency and along lines of human migration but in so doing it may miss places on the

route of its march The reason of this is not clear Certainly

Another point which is by no means understood is the fact that the disease remains for years in the endemic region and then suddenly spreads in epidemic and pandemic form extending at times over the whole world

The aetiology may, therefore be summarized by saying that the

is transmitted by food or insects and will be

in the lumen the glands the  
the small intestine probably  
which is set free and causes the

symptoms

obtained a virulent endotoxin In 1907 Strong carefully investigated the subject, and concluded that he was unable to find a soluble toxin as described above but that MacFadyen's endotoxin was the true toxin which caused the symptoms in man This endo-

causes the intestinal disturbance leading to the

leucocytosis  
leucocytes  
decreased  
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leucocytes  
decreased

the blood may be very marked  
and Shaklu gave great import-

## PATHOLOGY

The concentration of the blood causes a fall in the blood pressure which is indicated by the feeble compressible pulse but which to be studied properly requires investigation by means of a Riva-Rocci's instrument as has been done by Rogers and Megaw who found that in typical collapse the blood pressure might be only 60 to 50 millimetres of mercury when the patient is markedly cyanosed and restless. If the case is not so serious the pressure may be higher—70 to 80 millimetres of mercury—and if the patient is on the road to recovery it may reach to 90 millimetres. As a result of this low blood pressure the urine may be suppressed or scanty with a high specific gravity albumen casts and a large quantity of indican.

The body appears to react to the disease by the pouring out of substances from the blood which are probably bactericidal though Edwards's attempt to prove this failed because of decomposition and the admixture of other micro-organisms. Agglutinins are absent in fatal cases but according to Greig in non-fatal cases they begin to rise after the sixth day but drop after the twentieth day.

When recovery is about to take place the specific gravity of the blood decreases the blood pressure rises and the urine becomes abundant. As convalescence continues the great danger is secondary infection of the body by other micro-organisms which may cause serious illness and even death.

It is important to note that Greig has found the germ in the gall bladder (40-70 per cent of cases)—where it causes cholecystitis and in animals may give rise to gall stones—in the lung kidney and urine. According to Violle's researches bile in vivo tends however to hinder the development of the cholera vibrio. According to the same author a small dose of cholera toxin excites the secretion of bile while a large dose stops it. It is suggested that a septicæmia takes place but this has not been demonstrated. Manson suggests that it may pass by the lymph channels.

Danzys regards the disease as of anaphylactic origin.

**Morbid Anatomy**—Usually post mortem rigidity is very marked and the body keeps warm for some time after death. On cutting into the tissues it is noticed that they are very dry and that the blood is often thick and tarry. On opening the peritoneal cavity the hand experiences a peculiar sticky sensation felt in any other disease with which we are acquainted.

The stomach is usually empty and the bowels are reddish appearance with injected vessels. When a piece of small bowel opened the contents are usually found to be whitish or greenish grumous material more rarely will the contents be blood stained. These contents consist of food particles epithelial cells and white corpuscles and micro-organisms. When allowed to they separate into liquid and solid portions the former containing albumen and having a specific gravity varying from 1.005 to 1.015. The mucosa of the stomach and bowels is hyperemic and studded by ecchymoses. There is usually some



ment of the solitary and agminated glands as well as of the mesenteric glands

The liver is generally congested and the spleen is shrunken

with tubules

cloudy swollen

venous system full of blood while the arterial system and the left heart is empty. The lungs are collapsed, dry, and anæmic and the brain may be congested.

Microscopically the vibrios may be seen in Lieberkühn's follicles in the epithelial cells and in the mucosa of the intestine and the stomach. The kidney shows vascular congestion and destruction of the epithelium. Usually the vibrios do not penetrate into the blood stream and therefore the disease is mainly a general intoxication but Rebowski records cases in which they have been found in the liver, the kidney and the heart thus producing a general infection.

If the post mortem is held on a case which has died during the state of reaction pneumonia and the signs of other secondary infections may be found.

**Symptomatology**—A typical case of cholera has an incubation period which varies from a few hours to a few days (three to six). The onset is usually sudden but there may be prodromata in the form of diarrhoea or merely a feeling of illness and malaise. The attack begins with diarrhoea with or without colicky pains in the abdomen. The motions are at first feculent and contain bile but soon assume the typical rice-water appearance in which they are fluid and acholic containing numerous white flakes which when examined are found to be composed of mucus containing vibrios.

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place the patient passes into the algide stage in which the feature

of the circulation becomes more marked, the pulse almost disappearing at the wrist, the heart becoming weak and irregular, the respirations laboured, the skin cold and deeply cyanosed, the urine

with fluid again.

Sometimes convalescence is rapid. In some a secondary febrile condition ensues which may last for several days or even for a couple of weeks.

The patient may at times pass into a *status typhosus*, with a dry brown tongue, low muttering tremblings and toxic rashes, or or hæmorrhagic. The urine has a low specific gravity and contains are somewhat like those of

is stage from complications, or recovery may ensue after a long convalescence. The infection of the convalescent may be said to have ceased when on three separate days the bacteriological examination of the motions is negative.

The typical course of the disease as described above, is often (1) The stage of evacuation of reaction, which presents many varied features, in which, without any aid from the fæces. These

ready described comes rapidly followed by vomiting can appear. The post mortem and the bacteriological

examination of the bowel contents reveal the true nature of the disorder

pregnant women always abort and the fœtus may show signs of the disease. The reason of this abortive tendency is according to Schutz because cholera has a powerful effect upon even the non-pregnant uterus causing hæmorrhage during the stage characterized by muscular cramps.

**Sequelæ**—After such a severe illness it is usual to find more or less of the following sequelæ:—  
 1. Anæmia  
 2. Corneal hæmorrhage  
 3. Diarrhœa

It may be sporadic.

So closely may cholera be simulated by ptomaine poisoning, as

Ziehl carbolic fuchsin (1 in 50) for ten minutes or with Löffler's blue five minutes. If a large number of curved rods be present like those seen in

2. Inoculate four tubes of peptone water with the suspected stools the first with  $\frac{1}{4}$  c.c. the second with  $\frac{1}{2}$  c.c. the third with 1 c.c. and the fourth with 2 c.c. or inoculate each of two Erlenmeyer flasks capped with sterile filter paper without wool plugs with 1 c.c. of the stools. The formation of a scum on the surface of the medium within eight to ten hours is suspicious of cholera. The pellicle and the upper portions of the medium should be examined microscopically for the presence of vibrios and a microscopical agglutination test carried out mixing one loopful of the culture with one loopful of diluted cholera serum (1 in 1000). The peptone water should be tested for the presence of indol adding a

and MacConkey agar plates should be made and any suspicious

colony developing should be further examined and the germ

in stools which is agglutinated by this serum in a dilution not less than 1 in 2 000 can safely be considered as a rule to be the true germ of cholera. In doubtful cases all the cultural characters should be studied and Pfeiffer's test and Castellani's absorption test should be carried out.

3. Take a rice bke flake and smear it direct on to the surface of MacConkey's lactose agar plate using a sterile bent glass rod or Kruse's platinum pencil. Inoculate with the same rod or pencil

ards  
i  
the  
there is no zone of liquefaction in the medium cholera may be practically excluded. If there is liquefaction this may be due to the presence of the cholera or other serum liquefying germs or to the

6. *Bands Method*—The suspected fecal matter is inoculated into a

7. *Ottolenghi's Method*—The suspected stools are inoculated in a medium consisting of pure bile mixed with 3 per cent of a 10 per cent solution of sodium carbonate which after incubation at 37° C. for some hours is plated and further investigated.

8. *Ironson's Method*—This is an alkaline agar medium containing cane sugar and dextrin with fuchsin and sodium sulphite as indicator. Good results have been recorded by several observers.

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A modification of the method consists in inoculating ordinary peptone water tubes with the faecal matter and after four to six hours from the upper portions of these tubes inoculations are made in peptone water tubes containing a few drops of intestinal bacteria polyserums

If material has to be sent to a central depot for bacteriological examination certain precautions must be carried out The faeces

to state that no antiseptic must be mixed with the specimens beginning of the  
tubes The case  
50 per cent

**Treatment**—The treatment of cholera must aim at the destruction and removal of the vibrios the neutralization of the toxins the prevention of secondary infection through the damaged intestinal mucosa the healing of which must be assisted and the relief of symptoms

To promote these objects the patient must at once be sent to bed no matter how slight the attack may seem to be His room

The best treatment is that devised by Rogers which is as follows —

The patient is given as much calcium permanganate water (1 to 6

of the second twenty four hours eight pills are administered with in  
four hours and in severe cases this is repeated at the beginning of  
the third twenty four hours. In mild cases after the first twenty  
four hours the pills are only administered every four hours

### TREATMENT

The composition of the pills is —

Potassium permanganate 2 grains  
Kaolin and vaseline as may be required to make a pill

This pill is coated with a varnish composed of 1 part of salol and 5 parts of sandarach varnish or with keratin Pills kept for any length of time are apt to become hard and useless

The patient must be kept warm and supplied with plenty of water to drink which preferably should be taken in sips and sinapisms or turpentine stupes should be applied to the abdomen When collapse has set in hot bottles must be applied to the extremities and round the body and when the pulse fails the median cephalic vein should be opened at the elbow and Rogers's special silver cannula made by Messrs Down and Company inserted and through this injections of hypertonic saline solution should be injected until the blood pressure returns as tested by the pulse or more preferably by a Riva Rocci's instrument If the blood pressure sinks below 70 millimetres Rogers considers this to indicate the presence of a dangerous degree of collapse and an indication for an immediate intravenous injection He takes the specific gravity of the blood by the simple method of placing a drop of blood in the centre of a small vial containing a mixture of glycerine and water of known specific gravity (at the mean temperature of 67° gravity rises or falls trying another vial of lesser gravity is reached

blood pressure is 10 —  
administered If the specific gravity is over 1060 while the pressure is over 70 millimetres it is advisable to give an intravenous injection as the blood is dangerously concentrated and a single evacuation may lead to a rapidly fatal collapse

Restless  
intravenous  
is black

The hypertonic saline is —

- Sodium chloride
- Calcium chloride
- Potassium chloride
- Water

120 grains (8 grammes)  
25 grammes  
To 1 pint (575 c.c.)

The temperature of the sterilized solution (in an emergency 1 water filtered through cotton wool and boiled for fifteen min will suffice) has to be judged by the rectal temperature If the temperature of the body (99° F) or over Rogers injects the fluid at about the normal temperature of the body (99° F) — i.e. the risk temperature is 100° F If the rectal temperature is 1 degree or so below 99° F temperature of the solution in the flask should vary from 100° F to 104° F If the rectal temperature is high — i.e. over 100° F solution should be run in below 99° F if the temperature of the solution should not be warmed About 4 pints

are required for an adult male, but the case must be carefully watched, and the injection stopped if any distress of increased frequency of respiration is noted. These injections may have to be

given and warm rectal injections

During this treatment the bladder must be carefully watched to see whether urine is being passed or not and if suppression occurs dry-cupping should be performed over each loin.

sodium chloride solution) may be used

With regard to symptoms the persistent vomiting may be relieved by small pieces of ice or by  $\frac{1}{2}$  grain of cocaine dissolved in a teaspoonful of water or by a dose of 10 minims of *mistura pepsini composita et bismutho* every half hour until four doses have been given or by one or two drops of tincture of iodine in water. Cramps are treated by massage assisted by rubbing with dry powdered ginger, by hypodermic injections of morphia or by inhalations of a mixture of chloroform and oxygen. Prostration must be combated by hypodermic injections of strychnine or of camphor in ether. Delirium must be relieved by bromides with tincture of *hyoscyamus*.

The treatment of complications such as pneumonia etc must be conducted on the lines laid down in textbooks on general medicine.

Other methods of treatment may be briefly mentioned. Many disinfectants are recommended by different authors—e.g. a mixture of sulphocarbolates

drops of tincture of opium and warmed to 100° F. and given every four or five hours. Demerol's serum treatment has been found useless by

Strong  
50 c c  
Salm  
salm  
t have

With regard to diet no food should be given during the acute attack but merely liquids which should consist of water iced water iced soda water. Stimulants should only be given with

cardiac stimulant

When reaction sets in only the mildest foods must be allowed and then only with care. Begin with thin arrowroot and continue with milk mixed with soda water and then with milk barley water rice water etc. Sanatogen plasmon and somatose are also recommended. Meat extracts should be avoided. As improvement continues the diet may be slowly and carefully increased but the greatest care must be taken for a long time.

**Prophylaxis**—The prophylaxis must be based upon the knowledge that the disease is carried by man and is spread from one

**Private Methods**—At the beginning of an epidemic it is as well

compound are kept in a clean sanitary condition. He must also see that the filters are properly cleaned and should flies abound in the house the source should be diligently sought for or if it cannot be found and dealt with the Sanitary Authority should be informed. He should also see that the whole household keep themselves strictly clean especially the cooks.

With regard to food and drink care should be taken that all cooking

water as

or carbol

is safe

which in

cleaned with hot soda and water and exposed to the sun at least once a week. Food must not be stored near latrines and must be protected from flies and ants and the rooms and cupboards in which it is placed must be thoroughly cleaned with soda and hot water at least once a week.

All water must be boiled and filtered and stored in covered vessels and all milk must be boiled and carefully protected from



flies and other insects Care should be taken that good milk is procured No uncooked vegetables or salads should be used, fruit must be sparingly indulged in and unripe fruit, especially melons must be avoided Balfour advises that jellies in particular should and lime drinks

t be promptly treated by a medical man

With regard to prophylactics eucalyptus oil in 10 minim doses twice daily has been strongly recommended by some authors but the most usual prophylactic is a protective inoculation which was first introduced by Ferran in Spain and has been studied and improved by Haffkine and Gamaleia, by Tamancheff who added 0.5 per cent carbolic acid to the sterilized prophylactic, and by Strong and others

*Vaccines*—Haffkine originally used two prophylactics—a weak and a strong—with the idea that the strong would produce too violent a local reaction but this proving to be wrong only the strong is now used This prophylactic is prepared by intensifying the virulence of the vi

and then growing

sterile broth which

hypodermically into the flank as a dose There is some local reaction in the form of redness swelling and pain and some general febrile reaction The result is that after an initial diminution of

vaccinated there were 198 cases and 124 deaths and in 5778 vaccinated there were 27 cases and 14 deaths Vaccination confers a partial immunity which is said to last about fourteen months after which it diminishes and finally disappears Re

local reaction and but slight general reaction and increases the bactericidal and agglutinative powers of the serum considerably Strong considers that it contains receptors separated from the vibrios and that it probably acts by increasing the bactericidal and antitoxic powers of the epithelial cells of the mucosa of the alimentary canal

## PROPHYLAXIS

One of us has prepared an attenuated live vaccine by heating emulsions of agar cultures to  $45^{\circ}\text{C}$  or  $48^{\circ}\text{C}$  for one hour. A nucleo proteid vaccine according to Lustig and Galeotti's method can also be prepared.

*Tetrapaccine* (T A B C)—This is a vaccine prepared and used by Castellani since 1909 and now frequently employed in various countries as a prophylactic measure against cholera as well as typhoid and the paratyphoid fevers. It has been adopted in the Serbian Army since 1915. It is prepared as follows—

The growth of typhoid cultures is washed off with sterile 0.85 per cent salt solution to which 0.5 per cent carbolic acid has been added. The emulsion so obtained is stored at room temperature ( $18^{\circ}$  to  $20^{\circ}\text{C}$ ) for twenty-four hours and then standardized. To standardize it the germs are counted by using a Thoma Zeiss apparatus and sufficient carbolic salt solution is added to bring the number of germs down to 2,000 millions per cubic centimetre. The standardized emulsion is tested for sterility. The same procedure is carried out with paratyphoid A and paratyphoid B cultures. These two emulsions being also standardized to contain 1,000 million germs per cubic centimetre. The above procedure is also carried out with cholera. The emulsion of which however is standardized to contain 4,000 million germs per cubic centimetre. The four standardized emulsions when found sterile are mixed together in equal proportions and the vaccine will therefore contain per cubic centimetre—

Typhoid  
Paratyphoid A  
Paratyphoid B  
Cholera

500 millions  
250  
25  
000

Of this mixture 0.5 to 0.6 c.c.m. are given under the skin of arm or better into the loose tissue below the angle of the scapula the first time and double the amount a week later. A third dose of 0.5 c.c.m. given two weeks after the first is of advantage but essential for practical purposes. The amount of agglutinin each germ is about the same as if monovalent vaccine had been injected. The protection for cholera seems to last for about 3 months.

Castellani has prepared also a glycerol-tetrapaccine containing 2 per cent pure glycerine and standardized as to contain per c.c. typhoid 2,000 million, paratyphoid A 100 million, paratyphoid B 1,000 million, and cholera 4,000 million. Of this vaccine only one inoculation of 1 c.c. is given.

A pentapaccine having in addition 300 million of *B. pestis* is also prepared. The effect of the vaccine in man lasts for 6 months (six to seven).

**Public Prophylaxis**—It is the duty of the State to ward off cholera by preventing human beings from introducing the disease. This involves the careful watching of the frontiers especially the lines of intercommunication whether roads, railways, or ships. Under the last heading must be included ship

rafts for it must be remembered that cholera is very apt to be introduced by persons travelling along rivers

Any suspicious case must be detained for five days in quarantine in suitable isolation hospitals erected near the frontiers, while the sick must be tended in special hospitals with all the precautions to be mentioned later

Merchandise does not as a rule require any disinfection unless there is reason to suspect that some of it has been faecally contaminated from a case

When an epidemic begins the first duty is to form a special

the control of the epidemic } All suspicious cases must be reported to this central authority at once

Then central and outlying bacteriological stations must be provided and special hospitals and isolation hospitals built and if

public informed of the necessity of availing themselves of these medical arrangements If necessary a house to house inspection should be made in order to find out if there are mild concealed cases and nobody should be buried without a proper medical certificate

All patients must be removed to the hospitals and the houses disinfected with the Clayton gas apparatus in order to kill not merely the germs but also the flies and ants If this is not available then the use of lime may be used attending the the hands and

The dejecta of patients should be carefully disinfected with cyan or carbolic acid and no patient should be liberated from the hospital in three successive recalculation because a period of about six weeks at least

Care must be taken to disinfect and bury the dead with least chance of the infection spreading Cremation should be encouraged in preference to burial

A systematic search must be made for the origin of the infection and drinking water well water etc must be regularly examined bacteriologically Dangerous wells must be closed and all wells may be Hankenized—i.e. disinfected with permanganate of potash

The milk, ice, and aerated waters should be taken under the municipal control, and not merely must care be taken that they are pure, but they must be tested bacteriologically from time to time.

Vegetables must be inspected, and the place where they are grown ascertained and inspected, in order to find whether there is faecal pollution. A crusade must be made against flies and dirt in general. The disposal of sewage should of course have been dealt with before the epidemic has occurred, but if it is defective, attempts should be made to remedy this as far as possible, and a scheme at

allowed near wells. Drugs must be given free, and means for inoculation of the prophylactic provided on a large scale.

When the epidemic is past, the sanitary defects found out during its course should be remedied and not forgotten until another outbreak occurs.

#### SUMMARY OF PROPHYLACTIC MEASURES

##### *Public Prophylaxis* —

- 1. Protection of the frontiers by regular inspection posts and

#### PARACHOLERA.

*Synonym.*—\ dask (Senegal)

*Definition.*—Paracholera a term first introduced by Castellani



Therefore of dysenteric origin. Recently *Vibrio* has also found  
 id at times  
 the symptoms may be severe  
 The patient passes a number of serous motions those of  
 cholera. There may be algidity, cramps and the illness may  
 terminate in death. In most cases however the motions are now  
 and then tinged with blood which arouses suspicion as to its  
 dysenteric nature. Microscopical examination of a fresh prepara-  
 tion of the *faeces* may show a few red cells and leucocytes instead  
 of the epithelial desquamation found in true cholera. Bacterio-  
 logical examination demonstrates the presence of dysentery bacilli  
 and the absence of cholera and paracholera vibrios.

### Malarial Pseudocholera

This type is not rare in the tropics. The onset is sudden with  
 algidity and other symptoms closely  
 resembling cholera.  
 examination  
*faeces* are also useful in demonstrating parasites and the absence of cholera and paracholera.  
 Intramuscular injections of quinine quickly cures this type of  
 choleraic diarrhoea.

### Pseudocholera caused by Poisons

This is most usually caused by *arsenic* which is commonly in use  
 in the tropics especially in Ceylon where it can be bought in the  
 local markets. The diagnosis may be established by the history  
 and by chemical analysis of the vomit and *faeces* and negative  
 bacteriological examination.

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## CHAPTER LXXVIII

### THE DYSENTERIES

The term dysentery —Dysentery and dysenteric diarrhoea—Dysenteries caused by animal parasites—Protozoal dysenteries—Amœbic dysen

#### References

**The Term 'Dysentery'**—The term dysentery is derived from *δυσεντερία* signifying a bowel trouble and was first employed by Hippocrates. As used at present it covers a large number of distinct affections induced by various species of animal and vegetal parasites.

*Tenesme* and  
*Tormina* etc.

are too numerous in the Sinhalese term *Lehedanpachanas* (*Leh*—blood *hedan* mucus *pachanas* = diarrhoea) and the Indian term *Rattam seedam banthala porado* or *Wayatholechell*.

**Dysentery and Dysenteric Diarrhoea**—The occurrence of a bowel

chronic variety was called *radhisa*.

Hippocrates recognized two distinct types of disease of the bowels—one characterized by the number and fluidity of the motions which he called *δ'σποσα* and the other by the presence of blood in the motions which he termed *δυσεντερία*.

At first the term dysentery included any disease in which there

the disease was due to an ulcerated condition of the bowels and the contagious nature of the malady was early recognized and it was thought that its epidemics were due to miasmata. According to these ancient writers in order to make the diagnosis of dysentery

is only one phase of the clinical appearances of a dysentery, and is etiologically in no way different from what might be termed a true dysentery except in the severity of the symptoms and the post mortem appearances. Briefly, dysenteric diarrhoeas are dysentery under a somewhat different clinical aspect—viz., the stools do not contain blood and mucus, although the complaint is due to the same organisms as true dysentery. Hence it is not expedient to attempt to classify dysenteries by their clinical symptoms or by their post mortem appearances, and any rational classification and description of the complaint must be based on the causation.

We therefore classify dysenteries and dysenteric diarrhoeas into —

A Dysenteries caused by Animal Parasites —

- I " " " "
- II " " " "
- III " " " "
- IV " " " "

B Dysenteries caused by Bacteria

C Pseudo-dysenteries.

By the last expression we mean such forms of the complaint as may be due to the irritation of fish bones, glass cancer, or inflammations of organs adjoining the alimentary canal.

A. DYSENTERIES CAUSED BY ANIMAL PARASITES.

I THE PROTOZOAL DYSENTERIES

The protozoal dysenteries are classified into —

- (a) Amœbic Dysentery
- (b) Laveranic Dysentery
- (c) Leishmanic Dysentery
- (d) Ciliar Dysenteries.
- (e) Balantidic Dysentery
- (f) Sporozoitic Dysentery
- (g) Entoplasmic Dysentery

With regard to the term amœbic dysentery we prefer this term as less likely to be altered rather than the more correct nomenclature Loeschial dysentery or Loeschiasis.

(a) Amœbic Dysentery.

Synonyms —

b

F

F

**Definition.**—Amœbic dysentery is an acute or chronic specific disease of the intestine, caused most commonly by *Loeschia histolytica* Schaudinn, 1903, and possibly by other species. These amœbæ enter the body with food or water, and produce colitis, rectitis, and enteritis, characterized by the passage of frequent motions, which generally contain blood and mucus, and are associated with abdominal pain and tenesmus. At times they also

in the motions of a Lewis found the same organisms in the motions of cholera patients, while Loesch in 1875 which he found in the c diarrhoea Loesch's n which we call now at solutions of quinine of a strength of 1 in 5 000, when injected *per rectum*, temporarily benefited his patient, who, however, subsequently died of pneumonia, when a post mortem revealed that the bowels were ulcerated. Loesch was able to infect dogs, but on the other hand

introduced the term amœbic dysentery and Quincke, Roos Vivaldi, and many others published experimental researches on the infection of animals by amœbæ and the production of dysentery.

In the meanwhile a prolonged discussion took place some observers denying and others asserting the pathogenicity of the amœbæ. Kruse and Pasquale were the first to throw light upon

of Jurgens, who showed that there were two forms of amœbæ quite distinct from one another—viz, a *Loeschia coli* Loesch, which was harmless and another, *L. histolytica* Schaudinn, which was the true cause of entamœbic dysentery.

During recent years the labours of Hartmann, Whatmore, Wenyon, Fantham and Porter, Dobell and others have proved that the amœba which most commonly causes dysentery is *L. histolytica* and that the other forms of amœbæ described in dysentery by Viereck (*L. tetragena*) and other observers are different stages of this amœba, the cysts of which can be carried by domestic flies.

**Climatology.**—Amœbic dysentery is found throughout the tropical world, and also occurs in the Temperate Zone. In general terms it may be stated that amœbic dysentery is common in

Sporadic indigenous cases have been reported also from Great Britain where carriers of *Loeschia histolytica* cysts are not very rare especially among miners.

Our observations do not lend support to the theory that amœbic dysentery is more common in the hills than in the plains of the

far as they are known have already been described in Chapter XVII, p. 285. The most common source of infection is the drinking water contaminated by actual human fecal contamination.

Woodcock has called attention to the importance of a hot, damp climate as a factor in the spread of amœbic dysentery.

can retain their vitality for more than a fortnight in water.

There does not appear to be any well established racial or age predisposition, but the disease is less common in women than in men perhaps because they are in some way less exposed to infection.

**Pathology.**—The spores of the amœbe enter the body by the drinking water, and by food contaminated with cysts, often deposited by flies, and produce the young amœbe on arrival

probably by passing between the cells lining Lieberkühn's follicles and then entering the lymphatics make their way through the muscularis mucosæ into the submucosa where they live and feed upon the tissue cells red cells and perhaps leucocytes. They however invade not merely the tissue of the submucosa but also the radicles of the portal vein and at times the branches of the mesenteric arteries in which they may cause thrombosis. From the radicles of the portal vein they may be carried to the liver and cause hepatitis and hepatic abscess.

In the submucosa they induce cellular and œdematous infiltrations which cause the mucosa to project in the form of small

ulcers become infected with bacteria and quickly extend by the joint action of the bacteria and the amœbæ forming roundish or oval ulcers with undermined edges in the latter case the long axis of the ulcer lies transverse to the direction of the bowel. These ulcers may deepen until the muscular and the peritoneal coats are exposed and even perforated which of course leads to peritonitis or abscess formation according to the position of the perforation.

Amœbic dysentery would appear to undergo in many cases no spontaneous cure but may at times remain quiescent forming a scar which they

which is often black in colour from the action of the sulphuretted hydrogen of the bowel upon the iron of the blood. When cicatrization takes place the lumen of the bowel may be constricted causing stenosis and obstinate constipation. Peritonic adhesions are also very common binding the large bowel to the viscera or walls of the abdomen and pelvis.

Sometimes when the infection is severe the bowel becomes gangrenous at other times the amœbæ may be carried to the liver or other parts of the body and form abscesses which though most commonly met with in the liver may still occur in the spleen the salivary glands and elsewhere.

**Morbid Anatomy**—Usually the body of a person dying from amœbic dysentery is emaciated and the abdomen is sunken. Rigor mortis begins and passes off early and decomposition sets

be gangrenous in places or perforation and purulent congested and œdematous various organs may be note

enlarged and hyperæmic. The colon may be found adherent to the liver the spleen or the wall of the pelvis while internally the coats will be found to be reddened and inflamed and to show more or less numerous areas of ulceration and infiltration. These areas are most commonly found in the cæcum the hepatic flexure and the sigmoid colon but may occur anywhere along the course of the large intestine. In places small nodules surrounded by a ring of dilated vessels may be observed between which the mucosa may be deep ulcerated while the nodules may show superficial or deep ulceration. Deeper circular or oval ulcers may be noted with their surface covered with a dark reddish slough their edges under

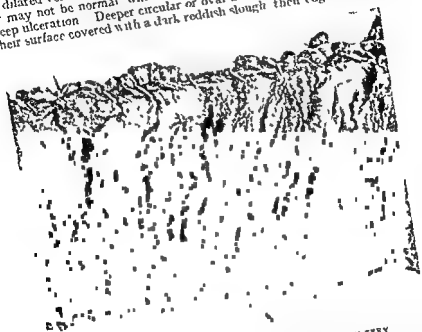


FIG. 10.—THE COLON IN A CASE OF AMEBIC DYSENTERY

microscopically and their base formed by the muscular coats. In the case of the oval ulcers the longer diameter lies transverse to the long diameter of the bowel as a rule. The peritonæal coat may be normal inflamed or softened and gangrenous. Usually the muscular coat is thickened as are the remains of the submucosa. Scrapings from these ulcers reveal all kinds of cells leucocytes bacteria and amebæ. The small intestine may show small bright nodules and the Peyer's patches may be enlarged. The vermiform appendix is usually normal but occasionally may be ulcerated. The liver is often fatty but may be congested and inflamed. The pancreas is generally normal but may show one or more abscesses.

but may be cirrhotic in chronic cases. The spleen is also usually normal but may contain an abscess. The kidneys often show signs of parenchymatous inflammation. The heart and lungs are generally normal but the former may be fatty or show brown atrophy and the latter especially the right lung may show a hepato pulmonary abscess.

**Symptomatology**—The symptomatology of amœbic dysentery may be classified into —

- 1 Acute Type
- 2 Chronic Type
- 3 Latent Type
- 4 Mixed Type

1 *Acute Type*—The onset is abrupt but may occasionally be preceded for a few days by slight diarrhœa alternating with constipation. Pain is felt in the lower part of the abdomen which may become very severe while the motions are attended with much griping and straining. These motions which rarely exceed thirty per diem contain blood and mucus and occasionally greenish material and when examined by the microscope reveal leucocytes mucus Charcot Leyden crystals amœbæ bacteria and at times shreds of tissue.

liver and spleen normal but tenderness is felt on pressure along the whole or a part of the large bowel.

The examination of the heart and lungs reveals as a rule no abnormality but the pulse and respirations are quickened. The microscopical examination of the blood usually shows that the red cells are diminished and sometimes that there is leucocytosis (upwards of 20 000 per cubic millimetre) and at times as first pointed out by Billet the number of eosinophiles is distinctly increased even when there is no concomitant helminthiasis.

The urine is diminished in quantity and sometimes contains albumen and casts but the skin is generally normal though there is often some fever of a remittent type which however may be entirely absent.

When the temperature falls to normal and the pain and tenderness

about the end of the first week and ten days from the commence  
by

ushered in by pain in the abdomen, tenesmus, and passage of blood and mucus in the motions, while the feces contain small greyish masses, in which the amœbæ may be found. The number of motions per diem is not excessive, and

dysentery may persist for years and cause the patient to slowly emaciate. The blood as shown by Chalmers and Archibald, often shows an increase in the large mononuclears.

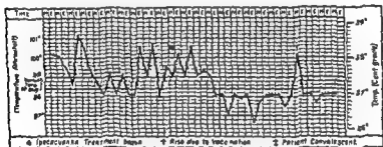


FIG. 763.—TEMPERATURE CHART OF A CASE OF AMOEBIC DYSENTERY COMPLICATED BY HEPATITIS.

easily lead to an acute attack, or to liver abscess, and no doubt these 'entamoebic carriers,' as has been noted by Martin, Vincent, and

Exhaustion may come on early, and the patient may die delirious or comatose, or, more rarely, from peritonitis, with or without perforation. More rarely improvement sets in, and the disease becomes chronic.



**Amœbic Fever or General Amœbiasis** — This term has been applied to certain cases of *L. histolytica* infections in which there is fever but no dysenteric symptoms and no sign of hepatitis. A few cysts are generally found in the stools and emetine induces a prompt disappearance of the fever.

**Complications** — The most usual complication is hepatitis and hepatic abscess, more rarely gangrene of the bowel and peritonitis may supervene during an attack while hæmorrhage is a most unusual complication. The hepatitis is recognized by the tenderness in the right hypochondrium and by the rise in the temperature but it and the hepatic abscess will be discussed in a subsequent chapter. In Ceylon we have observed cases of mixed infection dysentery and enteric, the two infections developing apparently contemporaneously.

**Sequelæ** — The important sequel to an attack of amœbic dysentery is liver abscess but abscesses in other parts of the body such as the spleen may be met with. Stenosis of the sigmoid colon due to cicatrization of the healing ulcers is well known and will be dealt with under the heading of Bacterial Dysentery. Sprue is often said to be a sequel of dysentery but this must be considered to be doubtful. Certain authors have described a persistent bradycardia.

**Diagnosis** — The diagnosis between bacterial and amœbic dysentery by clinical phenomena only is in our opinion impossible in most cases though several observers have attempted to define some differential points. These authors state that amœbic dysentery by its nature of toxic signs have in our experience no importance as we have come across extremely acute cases of amœbic dysentery with fever and toxic symptoms.

The only certain method of diagnosis is by the discovery of

*L. coli* and often contains erythrocytes which is the most important character. The nucleus of *L. histolytica* is eccentric, small and generally indistinct, does not stain deeply and has very little chromatin (see p. 313).

In order to expedite the diagnosis the French method is to add a drop of a 1 per cent solution of methylene blue to the mucus before placing on the coverslip when the pus and epithelial cells will be stained and the amœbæ more easily recognized as unstained clear objects.

The diagnosis of latent cases and of carriers is based on the finding of the characteristic histolytica cysts containing four nuclei. It is important to note that cysts are not often seen during the acute attacks with blood and mucus-pus.

In the search for cysts only a small quantity of material should be used well diluted with saline. The addition of a little iodine solution is useful as it makes the nuclei more visible. Dead cysts are easily stained by eosin

authors to be vegetal organisms—are more or less spherical 5 to 15 microns in diameter with a more delicate capsule than the cysts of Loeschke and enclose the cytoplasm to a narrow rim

structures described by Wenyon and varying between 6 and 10 microns in diameter and show frequently a iodophilic body which tends to be rounded or lobed

To facilitate the detection of cysts when these are in very small numbers various methods have been suggested though in practice as shown by Miss Porter such methods take a great deal of time and the results are not much better than those obtained by the simple immediate microscopical examination of several preparations. It

is of advantage to give the patient a saline purge, and then one of the so called enrichment methods of Cropper and others may be

Cysts maintain all their morphological characters for a very long period in faecal matter mixed with a formalin solution (2 per cent)

The presence in dysenteric stools of Charcot Leyden crystals as emphasized by Acton points to the condition being of amœbic origin rather than bacterial especially if there is scanty cellular exudate with preponderance of mono nuclears

**Prognosis.**—The prognosis in a case of amœbic dysentery must always be guarded, as there is the possibility of hepatic abscess, emetine and complete rest in the gangrenous cases, better in the acute, and still better in the mild chronic cases but the danger of latency after an apparent cure must be remembered. In the acute type hiccough is an unfavourable sign often indicating the approach of exhaustion and death.

**Treatment.**—It is of the utmost importance that the patient should be placed at rest in bed. For the same purpose the urine bottle and the bed pan must be used. It is advisable to relieve the severe griping by the use of opium, morphia or by the use of opium tincture of opium. The use of mucilage of acacia, codonopsis, or opodelme (gr.  $\frac{1}{2}$ ) suppository.

At first the bowels should be swept clean by a dose of castor oil (ʒi to ʒvi) with or without a few minims of liquor opii sedativus or a few doses of saline may be given during the first twenty four hours. After the castor oil has acted or

These drugs may be obtained in sterile tubes ready for injection. We have never seen any bad effects on the heart from the administration of emetine but we have met with cases of dermatitis probably due thereto while Dale and Low have noted diarrhœa after prolonged administration.

A combined subcutaneous and oral administration of emetine as recommended by Wenyon and O Connor (one grain emetin by injection in the morning and a  $\frac{1}{2}$  grain emetine tablet at night) answers well especially in subacute and chronic cases and in carriers.

If emetine cannot be obtained then ipecacuanha should be administered in 5 grain doses every three or six hours or in larger doses (gr x to xx) twice

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When the adult by

and as one other day of  
annic acid  
quinine in  
1 to 3 pint  
mass douche

should be used  
vessel by means of a long soft rectal tube well greased with hortic  
vaccine This injection may be preceded by a coeque or morphine  
(or  $\frac{1}{2}$ ) suppository introduced half an hour previously

— Acetozone (1 in 2000)  
or 1 in 1000) useful in

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With regard to the treatment of symptoms the relief of pain by hypodermic injections of morphia or by fomentations sprinkled with opium and applied to the abdomen as the case progresses favourably a bismuth mixture or tannalbin

## (g) Entoplasmic Dysentery.

In two cases of dysentery contracted apparently in Burma, Castellani

The protoplasm presents numerous roundish vacuoles none of which is contractile. No distinct nucleus is evident in fresh preparations. The parasite



FIG 766—*Entoplasma castellani*  
PAUL, 1914

Treatment.—Salines were administered with good effects. Emetine was also given but it is difficult to say whether it had any part in inducing the cure of the condition.

## II THE PLATYHELMINTHIC DYSENTERIES

*Fasciolopsis*  
teric symptoms  
the motions  
detail in Chapt  
detailed investigation

## III THE NEMATHELMINTHIC DYSENTERIES

specimens were submitted. It may be of course, that such organs were of such extremely delicate nature that they required the use of special methods

tion. The suggestion has been made that these bodies are probably forms of *Chilomastix mesnili* Wenyon, but the size of the bodies apart from other characters is evidently against this hypothesis.

and mucus

## IV THE ARTHROPODIC DYSENTERIES

## B. DYSENTERIES CAUSED BY BACTERIA.

## The Bacillary Dysenteries.

Synonyms.—*French* Dysenterie Bacillaire *Italian* Dissenteria Bacterica *German* Bacillenruhr

Definition.—The bacillary dysenteries are acute or chronic, endemic or epidemic, specific intestinal disorders caused by several

food or drink, more rarely by direct contact with contaminated

More rarely the bacilli cause a general septicæmia

History.—For years the theory of a bacterial causation for dysentery found many supporters, among whom may be mentioned Klebs, Prior, Ziegler, Hlava, Chantemesse, Widal, and Grigoriev, all of whom attempted to isolate a specific organism

Maggiora, Laveran, Arnaud, and Escherich believed that the

no gas in glucose media, and may not clot milk, and closely resemble Flexner's bacillus.

During an epidemic in the province of Oita, in Japan, Ogata isolated a bacillus which liquefied gelatine, stained by Gram's method, and produced intestinal ulcers in guinea pigs and cats a discovery which was confirmed by Vivaldi of Padua, but not by other observers

In the same year Calmette announced that the *Bacillus dys-*

a short bacillus not decolourized by Gram not clotting milk not

Kruse was also the first observer to state that there was more than one variety of *dysenteric bacilli*

In 1900 Flexner reported that he had isolated a moderately motile bacillus from cases of dysentery in Manila identical with

fermenting certain sugar media and could be distinguished from by the different biological tests These results have been frequently confirmed by observers in different parts of the world Strong isolated a bacillus slightly different from that of Flexner

made a detailed study of the pathogenesis of the disease

**Climatology** — Bacillary dysentery is found all over the world especially in the latter cause a type of the in factors in the infantile

mortality of those regions while they are also responsible for the endemic and epidemic cases so frequently met with in Europe and America

In the tropics they are also extremely common occurring more frequently at the end of the dry and the beginning of the wet seasons In certain regions and at certain times the virulence of the complaint appears to be increased the reason of which is not at present apparent Probably it depends upon the greater possibility of infection owing to the drinking water being highly contaminated in warm weather or perhaps to the presence of multitudes of flies The agency of these or other factors has not been completely inquired into Whatever the cause may be it is well known that at times the disease may spread in epidemic form over larger or smaller areas These epidemics may be institutional urban or rural or they may attack a district a country or a continent

to occur whenever sanitation is defective and hence is found prevalent in lunatic asylums and in armies in times of active service as is clearly shown by the present

## ETIOLOGY

war and this factor appears to be more potent than any climatological cause and indeed may be the principal reason why the disease is so prevalent in the tropics, though the high atmospheric temperature of these regions must assist the growth of the organisms

**Ætiology**—Bacillary dysentery is brought about by a group of closely allied bacilli which may be separated into several principal types according to their fermentative action on glucose mannite maltose saccharose lactose

- I **Shiga Kruse Type**—Glucose fermented (acid only) mannite, maltose saccharose lactose not fermented Agglutinated only by homologous serum Not agglutinated by normal horse serum Very toxic to rabbits
  - II **Flexner Type**—Glucose mannite maltose fermented (acid only) saccharose not fermented but there are exceptions lactose not fermented Agglutinated by homologous serum and Y serum and not unfrequently by Shiga serum and is shown by W Nicolle normal horse serum Non toxic to the rabbits
  - III **Strong Type**—Glucose mannite and saccharose fermented (acid only) maltose not fermented Agglutinated by homologous serum only Most authorities consider it non toxic but the original Strong strain was very toxic to rabbits
  - IV **Miss and Russell Type**—Glucose and mannite fermented (acid only) maltose saccharose lactose not fermented Agglutinated by homologous and Flexner serums at times by Shiga serum and normal horse serum
  - V **Flexner's dysentery (Casellani) Type**—Differs from all above groups in fermenting (slowly and acidity only) lactose in addition to glucose Well agglutinated by homologous serum Not agglutinated by Flexner and Shiga serum Non toxic to rabbits
- For more details on bacteria of the dysentery group the reader is referred to Chapter XXXVI p 96 Less important types are Ruffer and Willmore's *Bacillus F17* or No 1 Bacterium's *Bacillus dysenteriae* Shiga's *B. dysenteriae* Tokio Castellani's *Bacillus paradyentericus* etc
- Hiss divides the dysentery bacilli into 4 principal groups—
- Group 1 Ferments glucose only (Shiga Kruse bacillus)
  - Group 2 Ferments glucose and mannite (*Bacillus* 3)
  - Group 3 Ferments glucose mannite and saccharose (Flexner Mann bacillus)
  - Group 4 Ferments glucose mannite saccharose maltose and dextrine (Hirtz's bacillus Wollstein's bacillus)
- Hirtz's bacillus and Shiga have called attention to the fact that while fermenting mannite is a reliable means of differentiation not so in the case of mannite as the fermentation of maltose saccharose importance can be given to the fermentation of maltose saccharose dextrine
- Lehman and Neumann give the following classification
- 1 Shiga Kruse No fermentation of mannite maltose saccharose
  - 2 Flexner Ferments mannite and maltose
  - 3 Strong Ferments mannite has no action on maltose and saccharose
  - 4 B 3 Ferments mannite has no action on maltose and saccharose
- Hiss has noted that the various strains may vary their fermentative character when passing through flies



Under the term *B pseudo dysentericus*, Kruse described a non lactose fermenting bacillus which is the cause of many outbreaks of asylum dysentery in Germany. Later he used the term 'pseudo dysentery' to denote every variety of dysentery caused by bacilli other than those of the Shiga Kruse type.

Cells of *B coli dysentericus*, according to the original description, is motile, clots milk, and produces gas in some sugar media, and therefore cannot be considered to belong to the dysentery group. According, however, to more recent descriptions by De Blasi and others, some strains of the *B coli dysentericus* may not produce gas and may not clot milk, and closely resemble Flexner's bacillus.

The true dysenteric bacilli may be differentiated from one another by their varying fermentative actions upon carbohydrates, by agglutination, Pfeiffer's reaction, and Castellani's absorption method. A table showing the characters of the more important dysentery germs as well as other intestinal bacteria will be found in Chapter XXXVI, p 944. We wish to emphasise however, the fact that the biological reactions of the bacilli are much more reliable than the fermentative changes.

The dysenteric bacilli are distributed mainly by the faeces of persons suffering from the disease, but there are also 'dysentery carriers' in the true sense of the word, who are perhaps not merely an important source of infection but possibly the important factor in the dissemination of dysentery.

... takes place by  
of the dysentery  
sentry, charac-  
od and mucus, from which the  
infection is direct contact with a  
patient or a carrier or with articles or more especially food con-  
taminated by them. The bacilli are conveyed from faecal matter  
to the mouth by the contamination of food or drink or the utensils  
The method of infection is prob-

strated by one of us, are capable of carrying the bacilli and are therefore probably a prolific source of infection in tropical countries. Luckily the water supply is very rarely infected. With regard to lower animals, Kruse and Bowman have recorded spontaneous bacillary dysentery in monkeys, due to bacilli of the Flexner type. The method of infection is prob-  
a bacillus of Type Y is the  
that there may be a possi-  
rtance in the spread of the  
disease.

It would, however, appear that the bacilli are capable of living in the alimentary canal without causing the symptoms of the

## ETIOLOGY

disease until the vitality of the host is lowered by some agency, such as a chill or an attack of diarrhoea or some intercurrent disorder, when they are capable of producing their ill effects.

This carrier problem in the aetiology of dysentery is of great importance and though as yet it has never been conclusively proved that an outbreak has been due to a carrier still it is known that the bacilli can be excreted in an intermittent manner by people suffering from mild relapses. The dysentery carriers are classified into—

- (1) healthy
- (2) precocious
- (3) convalescent
- (4) relapsing and
- (5) chronic carriers.

The healthy carrier is rare but May has found 22 out of 573 persons examined during an epidemic and relapsing carriers are well known. The convalescent and precocious carriers are well known of which the latter are of the greatest importance in spreading the disease. The chronic carrier excretes but few bacilli but may be of importance in the spread of the disease. With regard to the geographical distribution of the principal forms of bacilli it may be stated that practically all of them seem to have a cosmopolitan distribution.

*Aetiological Classification of Bacterial Dysenteries*—An aetiological classification of bacterial dysenteries has been suggested as follows—

- I Due to *B. dysenteriae* Shiga kruse—*Bacterial dysentery sensu stricto*
- II Due to mannite fermenting dysentery bacilli (Flexner, Hiss and Russell Strong)—*Paradysenteries*
- III Due to germs having the general character of the dysentery bacilli but slowly fermenting (acidity only) lactose and not agglutinated by Shiga kruse and paradysentery serums—*Meladysenteries*

**Pathology**—The bacilli taken into the body with food and drink pass to the intestine in which they grow and multiply and also the whole length of which they can be found. The researches of Flexner and Sweet have proved that the bacilli can abound in small intestine where no pathological lesion may be found. The bowel they give rise to the toxins of which two are known one which acts upon the lower bowel and the other on the nervous system. Both these toxins are absorbed into the blood but first being excreted by the large bowel causes the lesions known to be associated with dysentery and explains the local causes an exudation of lymph into the submucosa and later the mucosa. This lymph coagulates and is invaded by a crenate and in due course the glands and the tissue of the crenate and the muscularis mucosae are destroyed by coagulative thrombosis of the vessels. This fibrinous or diphtheritic membrane is at first most marked on the summits of the ridges and may not be found at the bottom between the varying channels. Large numbers of micro-organisms of varying kinds of the submucosa may reveal accumulations.

leucocytes, and the peritoneal coat may be œdematous. The micro-organisms destroy the fibrinous false membrane, which may separate off in flakes, thus causing ulcers, which are at first superficial, but later become deep and extensive. After treatment these ulcers heal with the formation of connective tissue, thus producing a scar in the mucous membrane, which in due course becomes pigmented from the sulphuretted hydrogen of the bowel acting on the iron of the blood. The other toxin may attack the nervous system, causing peripheral neuritis.

Very rarely do the bacilli enter the blood stream, and cause  
 the  
 had

been prematurely expelled from the uterus of a mother who was suffering from bacillary dysentery. Darling has actually grown the bacillus from the blood of cases of bacterial dysentery. Occasionally the bacilli affect the joints and very rarely the conjunctiva.

**Morbid Anatomy.**—On opening the abdomen, the peritoneum is found in general to be normal, but the bloodvessels of the large bowel are seen to be injected, and the mesocolons may be infiltrated with lymph, or firm and fibrinous. There may be adhesions of the sigmoid colon to the omentum, pelvis, bladder, or small intestines, while the splenic flexure may be adherent to the spleen and surrounding parts, and the hepatic flexure to the liver. The cæcum may show adhesions to the omentum, and more rarely there may be pericæcal abscess. Signs of a general peritonitis may be met with, and will generally be associated with a gangrenous or perforated condition of the intestine.

The small bowel is usually normal, but may be hyperæmic or, much more rarely, ecchymotic. The walls of the large intestine are usually considerably thickened and hyperæmic, and may at times be found to be gangrenous along a great or lesser extent of their course. On opening the large bowel, the mucosa will be seen to be covered with a coagulated exudate in the form of a false membrane which is more evident on the summits of the folds,

ulcers may be very extensive, leading to the separation of large sloughs, or may extend deeply into the coats of the bowel, causing perforation and peritonitis or in less serious cases induce the exudation of much lymph into the peritoneum coat, which subsequently causes adhesions.

In post mortem examinations of cases which have died from  
 the intestine matted  
 and the  
 found

scarred by old dark-coloured cicatrices, indicating the position of the healed ulcers of a previous dysentery. More rarely the cicatrization may have proceeded to such an extent as to cause narrowing of the lumen of the gut, and still more rarely may the process lead to abscess formation in the adherent omentum the pus of the abscess slowly working its way into the anterior abdominal wall and so to the exterior. The cecum and other parts of the bowels may show polypi protruding from the mucous membrane, a condition often called *colitis polyposa*.

In the epidemic diarrhoea of infants, the lesions which may be found are classifiable into hyperplasia of the agminated and solitary glands, superficial ulcers lesions resembling those described above or invisible lesions.



FIG. 707.—THE COLON IN A CASE OF BACILLARY DYSENTERY

Microscopically, there is at first an exudation of fluid containing but few cells into the submucosa, while the mucosa is congested but the glands are seen to be quite normal. A little later the exudate into the submucosa is seen to have formed fibrin, and the vessels of this coat are noted to be dilated to contain numerous polymorphonuclear leucocytes which may be seen undergoing

submucosa. These sloughs may be small or large, and even at times may be tubular. The motions are now exceedingly numerous, and sometimes hæmorrhage *per anum* may take place. The patient becomes extremely exhausted, the pulse small and frequent, the temperature falls to subnormal, and the extremities become cold up to the knees and elbows, while the motions are passed involuntarily, and death takes place in some two or three days from exhaustion. Recovery from an attack of gangrenous dysentery is extremely unusual, but when it does occur the pulse slows, the temperature rises, the motions become fæculent again, and the urine

3  
and  
keeps high, assuming either a continuous or a high remittent type, and is associated with a dry tongue, and a mouth covered with sordes, fœtid breath, headache, malaise, pains in various parts of the body, marked epigastric disturbance, and occasionally with ecchymoses under the skin in various parts of the body. The characteristic signs of abdominal pain and tenesmus are absent, and the

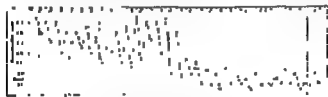


FIG 768.—TEMPERATURE CHART OF A CASE OF BACILLARY DYSENTERY, SHOWING THE EFFECT OF SERUM TREATMENT

Chart by Archibald

motions, though containing blood and mucus, are mixed with considerable quantities of fæculent matter. The mind is clouded, the patient becoming very stupid, sleepless, and at times delirious. Abscesses may now appear in varying parts of the body, but especially in the parotid or the ischio rectal fossæ while carbuncles, bedsores, and peritonitis may also occur. Death usually ensues after a few days' illness from exhaustion, toxæmia, or hyperpyrexia.

4 *Choleraic Dysentery*—Castellani called attention some years ago in the tropics, and again recently in various war zones, to a type of dysentery or serous diarrhœa which is often mistaken for cholera. The onset is sudden, with rice water like or serous motions, there may be vomiting, and the condition of the patient becomes rapidly very grave. In most cases, however, a motion is passed now and then tinged with blood, and this clears the diagnosis.

5 *Chronic Bacterial Dysentery*—Chronic dysentery appears after an attack of acute dysentery which has apparently been cured, but in which, after a period of quiescence, diarrhœa appears. About five or six motions, composed of watery, evil-smelling, fæcu-

# SYMPTOMATOLOGY—COMPLICATIONS

lent matter are passed per diem and may at times contain blood and mucus or simply mucus. After a time these symptoms disappear and the motions become normal or there may be slight constipation. In due course however the diarrhoea returns and these exacerbations recur for months and years and seriously affect the patient's general health. The appetite becomes bad the tongue red and smooth or flabby and coated digestion is impaired and fermentative changes cause the bowels to be distended with gas while hemorrhage slight or severe may take place from the nose or under the skin. Profuse sweats may occur at night and tend to exhaust the patient or the diarrhoea continuing fitfully transiently or after treatment patient who may die of exhaustion may gradually wear out the system. Some authorities believe that chronic dysentery may develop in a case of chronic dysentery or some intercurrent disease. We do not agree with them although of course sprue may develop in a case of chronic dysentery.

6 *Dysenteric Diarrhoea*—The term dysenteric diarrhoea may be applied to the non bloody diarrhoea of chronic dysentery and to those cases of diarrhoea which occur during an epidemic of dysentery and in which the bacillus is either proved to be present in the motions or the patient's blood agglutinates in high dilution one of the dysenteric bacilli. The attack may in no way differ from an ordinary attack of diarrhoea due to other causes but is apt to recur and to turn into chronic dysentery.

7 *Dysenteric Infantile Diarrhoea*—Diarrhoea is an extremely common disease among infants in the tropics but has not yet received the attention which it has in the United States in Europe and in Japan where it is called *Chari*. It is believed to be due to Flexner's bacillus or more rarely to Shiga Kruse's bacillus while the symptoms resemble enteric dysentery being characterized by beginning with vomiting and a rise of temperature from 103° to 104° F a dry mouth coated tongue distended and tender abdomen and the passage of motions containing feculent matter often green in colour generally mixed with blood and mucus. The fever is the remittent type and as the disease progresses the child weakens and may become convulsed or comatose and die or may live several weeks suffering from repeated attacks of diarrhoea and finally from exhaustion. If recovery is to take place the temperature declines and the diarrhoea ceases but the child is left in a protracted debilitated condition from which it takes months to recover.

*Complications*—Peripheral neuritis is not uncommonly met generally in a mild form and often confined to the nerve. Arthritis and polyarthritis are also not uncommon while inflammation of the tendon sheaths may also take place. In enteric dysentery pyrotid buboes are not uncommon while abscesses in other parts of the body and peritonitis may develop. In gangrenous dysentery hemorrhage may be a serious complication. Typhoid fever may occur at the same time as a dysenteric attack and is not infrequently met with several cases of ap



FIG 769—INTESTINAL SCHISTOSOMIASIS  
(After Symmers)  
Polypoid growths in descending colon

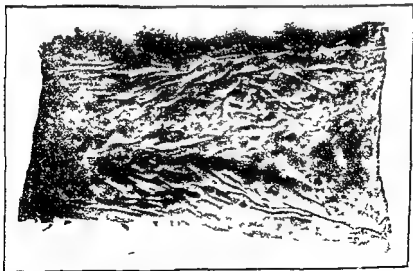


FIG 769A—BOWEL IN A CASE OF INTESTINAL SCHISTOSOMIASIS

## SYMPTOMATOLOGY

and cause dense cellular infiltrations giving rise to fistulae, which may spread and infect a large cutaneous area.

When the ova reach the liver by the blood stream they give rise to fibrous-tissue formation or an abscess. The surface of the liver shows in places a whitish new work and also flat chana white plaques. On section a marked increase in Giesson's capsule may be seen the portal vessels lying in circular or slightly oval areas of connective tissue. Gall stones may also form round the ova in the gall bladder.

The pancreas and spleen may also be infected and the vulva and vagina. The ova may also reach the lungs causing a chronic interstitial pneumonia and passing through these organs may enter the heart and the general circulation.

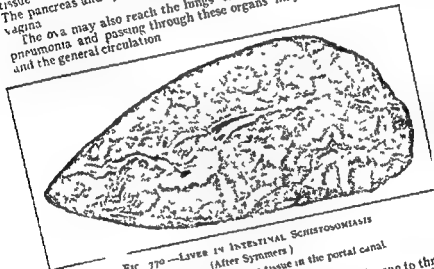


FIG 770—LIVER IN INTESTINAL SCHISTOSOMIASIS  
(After Symmers)

Shows the increase of fibrous tissue in the portal canal.

**Symptomatology**—The incubation period seems to be one to three months. The clinical appearances of the disease may be classified into four varieties—

- 1 Slight infections
- 2 Schistosomal dysentery
- 3 Schistosomal tumours
- 4 Schistosomal fever

**Slight Infections**—In these cases there are no symptoms and the parasite is discovered by the examination of the feces by a microscope.

**Schistosomal Dysentery**—The symptoms resemble those of cholera consisting of pains in the abdomen and the passage of blood and mucus. The attack begins with a characteristic frequent small motions containing the characteristic mucus. The result of these frequent motions is to cause pain.



**Ætiology.**—This is entirely unknown. The disease occurs in man and animals, fowls, dogs and calves, and is said to be very contagious. The Venezuelan natives say that it arises from children chewing the green tender stalks of unripe maize.

resents two pathological pictures—  
which is rare, and a low infection

convulsions may supervene, but in either case the patient dies.

**Diagnosis.**—In an endemic area severe dysenteric symptoms in a native child would lead one to suspect the disease.

**Prognosis.**—This is extremely bad, as the disease appears to be always fatal.

**Treatment.**—Natives treat the disease by enemata of strong

**Prophylaxis.**—Nothing can be said with regard to this until the disease is scientifically investigated and its ætiology discovered.

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**DIVISION II DISEASES OF THE SYSTEMS**

**RESPIRATORY SYSTEM**

**CIRCULATORY SYSTEM**

**LIVER AND PANCREAS**

**DUCTLESS GLANDS AND METABOLISM**

**URINARY SYSTEM**

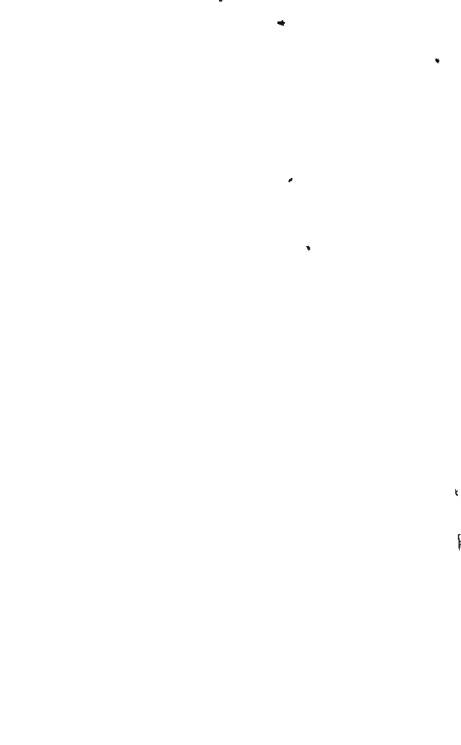
**GENERATIVE SYSTEM**

**LYMPHATIC SYSTEM**

**CONNECTIVE TISSUES MUSCLES BONES AND JOINTS**

**NERVOUS SYSTEM**

**ORGANS OF SPECIAL SENSE**



## CHAPTER LXXXI

# DISEASES OF THE RESPIRATORY ORGANS

General remarks—Rhinis spastica vasomotoria—Gangosa—Hirudiniasis—  
Rhinal cholerae—Linguatubiasis—Rhino pharyngitis spirochætica—  
Bronchial spirochætosus—Tropical bronchomycoses—Pulmonary  
nocardiasis—References

### GENERAL REMARKS.

DISEASES of the respiratory organs are of common occurrence

Liver abscess may burst into the lung or pleural cavity, and may cause the expectoration of a peculiarly brown and viscid sputum. *Emphysema* and *asthma* are fairly common and all types of bronchitis are met with though more rarely than in temperate zones.

*Phthisis* is common and appears to be increasing in the East, but it must be remembered that the pulmonary lesions of paragonimiasis, histoplasmosis, bronchospirochætosis and certain broncho-

### RHINITIS SPASTICA VASOMOTORIA.

Synonyms.—Rhinis nervosa, Coryza spasmodica, Dyspnæ tropica, Tropical hay fever.

Definition.—A rhinitis characterized by fits of sneezing with

**Symptomatology.**—The affection is characterized by attacking adults in whom it causes violent fits of sneezing lasting from a few minutes to two hours, during which time the victim sneezes ten to seventy times, while fluid pours from the nose, tears roll down from the eyes, the conjunctivæ are injected, the eyelids swollen, the head aches, and the patient is unable to do his work. The attacks are repeated constantly, and may take place several times during the day, or may not recur for weeks. They may be associated with or followed by dyspnoic conditions resembling to some extent asthma. At the beginning the mucosa of the inferior turbinated bone may appear hyperæmic, but later it becomes swollen, and has a macerated appearance. The disease therefore closely resembles that caused by dust acting on the mucous membrane of the nose, whose nervous system is especially sensitive in the tropics. We have seen it in the West Indies, where it is exposed to the dust of tea and copra.

**Treatment.**—The only successful treatment in many cases is a change of climate, when the symptoms stop at once. When the patient cannot have a change of climate atropine and strychnine pills or small doses of quinine may be recommended and locally a spray of a solution of cocaine ( $\frac{1}{2}$  or 1 per cent) and adrenalin (1-2 per cent).

### GANGOSA.

and larynx, destroying cartilage and bone, and causing much deformity.

**History.**—The disease appears to have been first described in 1828 by a Spanish Royal Commission to the Marianne or Ladrone Islands under the name 'gangosa, which means nasal voice,' and is derived from *gangoser*, 'to snuffle.' It was carefully studied by ... describes ... tion to the ... cGregor as ... the disease ... d by Leys ... ds and by ... In 1907 ... e from the ... t on a case ... and Kerr ... t it closely

ea for the

disease it being common in the Rat and

5

and Leys are opposed to it

the future

it appears to be equally common in males and females.

**Pathology**—It appears to begin sometimes as a coryza or forms with bone wit is due to

on the p <sup>apparently</sup> limited to a small infiltration some giant celled formation and proliferation of blood vessels with formation of granulation tissue

**Morbid Anatomy**—The post mortem may reveal signs of some concomitant affection—for example tuberculosis pleurisy or cardiac hypertrophy—which have nothing to do with the disease the important features of which

with another  
 sometimes there are giant cells and proliferating vessels and there are always hemorrhages. The surface of the ulcer at seen sending processes into

of large vacuolated cells with pale vesicular nuclei ; Some diplo cocci micrococci and bacilli have been noted but no acid fast bacilli or *Treponemata* have been seen

always first the soft parts and then the bone of the palate the nasal



FIG 772—GANGOSA  
(From a photograph by Arnold)

septum and the cartilages of the nose so that the skin falls in and the nose and mouth are converted into one cavity It may then extend on to the face or lip or affect the larynx When it spreads over the face it may involve the eyelids erode the cornea and even destroy the vision In some advanced cases the entire front of the face is replaced by a large opening ringed about by foul ulcers Sensation is diminished over the ulcer and a most objectionable odour is exhaled while a slight discharge of granular and necrotic debris is generally present The ulcers may also appear on the skin of the extremities or on parts of the body not usually covered

with the clothing. At times the ulcers may remain quite superficial spreading at one edge while healing at another. Scarring similar to that seen after burns may result which by contraction may lead to obliteration of the palpebral fissures, the nasal orifices and to reduce the size of the mouth as well as to produce great deformities in the hands and feet. It may cause a chronic osteitis resembling that seen in syphilis. It appears to be in some way self limited as it does not attack the trachea or genital organs. The ulceration may progress continuously for a period of ten to thirty five years or it may advance at certain times and be quiescent at others or it may cease at any time leaving a chronic ulcer. Its

**Variety.**—A fulminating variety has been described by Mink and McLean in children which is fatal in forty eight hours and closely resembles diphtheria without however the presence of the specific bacilli.

**Diagnosis.**—The diagnosis has been practically discussed in the **Etiology** and need not be repeated except to say that it must be made by a process of exclusion.

**Prognosis.**—<sup>72</sup>  
 mate recovery  
 great. The ge  
 may take plac  
 dysentery and other internal causes.

**Treatment.**—Mercury is useless and potassium iodide has little action on the condition. Salvarsan and neosalvarsan or their substitutes seem to give much better results. An application of a strong solution (1 per cent) of permanganate of potash is recommended as a deodorant and local application of tincture of iodine or the actual cautery.

**Prophylaxis.**—Nothing definite can be said under this heading, as the etiology is not known with certainty. But segregation of the patients in a colony or special hospital until they are cured is advisable. In Guam according to Angeney the disease is steadily decreasing thanks to the measures taken of segregating the patients and possibly to the thorough treatment of yaws patients and also probably to the improvement in general sanitation since the American occupation.



## HIRUDINIASIS

**Definition**—Hirudiniasis is the invasion of the nose mouth pharynx or larynx by leeches

**Remarks**—Leeches have already been described in Chapter

and Ceylon less so in the Philippine Islands Java Sumatra Australia and Japan

**Ætiology**—It is the water leech which lives in springs which is the cause of the trouble to man as it is apt to be swallowed with

mouth nose pharynx or larynx does not suck blood until gorged and then detaches itself from the affected part as is the rule when it attacks the skin but

is hardly likely that a leech though such an occurrence

**Symptomatology**—The patient usually knows that he has swallowed a leech and has felt the animal catch hold of the mucosa of the pharynx during the swallowing of the water. But children and even adults may be quite unaware of the accident having happened. The most important sign is the bleeding from the nose or mouth or the hawking up of blood from the pharynx or hæmoptysis from the larynx accompanied by a short irritating

with a nasal speculum or a laryngoscope make the diagnosis easy

**Prognosis**—This is usually good if the parasite is removed in time but if left for long removal may be too late and the patient succumb

**Treatment**—Apply a pledget of cotton wool soaked in 30 per cent of cocain to the parasite. This produces a paralyzing As however the might fall from the ke the patient lie prone on a couch with the head hanging over when the paralyzed parasite will be coughed up



ally the affection may run a much longer course and may spread to the larynx trachea and bronchi. In two cases of bronchitis following an attack of spirochætic coryza above described Castellani observed in the expectoration the same type of spirochæte and it would appear therefore that there may be several types of bronchial spirochætosis.

**Diagnosis**—This is based on the microscopical examination of the nasal and pharyngeal secretion. The beginner should be careful not to recognize as spirochætes detached cilia and segments of

bolitic acid 3 minims cocaine hydrochloride 1 grain water 1 ounce) will be found useful. Aspirin, pyramidon and quinine may be administered internally in 5 grain doses three or four times daily. In cases running a protracted course arsenic may be tried.

In a few cases of naso-pharyngitis preceding at times typical cases of bronchospirochætosis the nasal and pharyngeal secretion may contain *S. bronchialis* though this is rare. Several ætiological types of nasal spirochætosis might therefore be perhaps distinguished. In the tropics one comes across occasionally ulcerative affections of the nose with presence of numerous coarse spirochætes and *Bacillus fusiformis* but these conditions have nothing to do with true rhino-pharyngitis spirochætica.

## BRONCHIAL SPIROCHÆTOSIS

**Synonyms**—Castellani's bronchitis (Galli Valerio) Spirochæte bronchopulmonaire de Castellani (Violle) Bronchite sanglante (Violle)

**Definition**—A type of bronchitis and broncho-alveolitis characterized by the presence of enormous numbers of spirochætes in the expectoration.

**History**—The affection was described by Castellani in 1905  
*S. bronchialis* in 1907  
 by Branch in 1906  
 Jackson in 1908 in  
 ribed numerous cases  
 and Kilbourne a case  
 nberlain recorded two

further cases.

In 1913 Chalmers and O Farrell carried out an investigation on the malady in the Sudan and succeeded in reproducing it in

Galli Valerio recorded several interesting cases of the malady in Switzerland and Iurie one in Serbia. In 1917 Galli Valerio recorded further cases in Switzerland. In 1917 Violle first discovered the affection in France making a very complete investigation and publishing numerous papers on it. Violle's observations stimulated further research in France and a number of cases of the malady were reported by Bine Didi and Riberau by Letter by Dohmier by Barbary and others. Alcock has described a case in an English soldier in the North of Italy. Villa and later Corvetti have recorded cases in South America.

**Geographical**—The disease has probably a cosmopolitan distribution. It has been found in Ceylon India Philippine Islands China and Indo China North and Equatorial Africa being especially common in the Sudan West Indies America, and recently in Europe in the Balkans Italy Switzerland France and England.

**Ætiology** The disease is due to *S. bronchialis* Castellani 1907. The parasite has been further investigated by several observers and in a masterly manner in 1914 by Lantham who described its oral and intracellular stages. The organism is extremely polymorphous being very variable in length thickness and the number of waves. One may distinguish thick and thin forms long short and intermediate types. The length may vary between 5 and 3 microns its breadth between 0.2 and 0.6 micron. A number of the parasites are between 14 and 16 microns or 7 and 10 microns the latter resulting as shown by Lantham from transverse division of the former.

The ends are of variable shape but mostly somewhat acuminate.

The number of spirals varies between two and eight. Flagella seem to be absent but Lantham has noticed the presence of a delicate membrane or crusta in certain specimens.

In fresh preparations *S. bronchialis* is actively motile for only a short time the motile phase as demonstrated by Lantham is succeeded by one of granule formation the granules or coccoid bodies

in several features. *S. bronchialis* is more actively motile than the oral spirochetes it does as observed first by Chalmers and O'Leary and later by Taylor very quickly in fresh preparations while the oral spirochetes may live for hours outside the human mouth. Coccoid bodies are much more frequently produced by *S. bronchialis* than by the spirochetes of the mouth. Intracellular stages are occasionally seen in the case of *S. bronchialis* but not in the case of spirochetes from the mouth. *S. bronchialis* strains with more difficulty than the oral spirochetes is slenderer than one of them *S. fusca*, and does not appear to produce pseudo-membranes.

**Predisposing Causes**—A chill acts in our experience, as a very important predisposing or secondary cause.

**Experimental Reproduction**—Chalmers and O'Garrell have suc

persons the spray exhaled in coughing etc being contaminated with the spirochaetes or more probably according to Fanthar with the resistant coccoid bodies produced by *S. bronchialis*. It is also probable that a certain number of persons may harbour *S. bronchialis* and that a chill or an acute attack of the disease

usually violent and others have called attention

feels chilly and develops fever which generally is not very high (seen two and eight) have rheumatoid is scanty mucopurulent very seldom containing traces of blood. In most cases the general condition of the patient is not much affected in others the patient feels very tired and ill.

**Subacute Bronchospirochaetosis**—The attack begins suddenly or

take place. The physical examination of the chest may reveal patches and may sputum is

normal and so is the differential leucocytic count.

**Chronic Bronchospirochaetosis**—Chronic bronchial spirochaetosis may follow on an acute or subacute attack or several such attacks but frequently the onset is quite insidious and slow. The patient has a chronic cough which is in certain cases more severe in the morning. The expectoration is not very abundant and may be mucopurulent in character but in many cases for periods of two or three days and even much longer the expectoration contains blood. Sometimes attacks of genuine hæmoptysis occur one or two teaspoonfuls or much more of blood being expectorated. In some cases there is no fever in others a true hectic like fever may be present. In some cases however the rise of temperature takes place in the morning and not in the afternoon in others the fever is present only occasionally and is very irregular. The physical examination of the chest reveals in many cases very little except a few dry or coarse moist rales. In others there may be signs of consolidation. The general condition may remain fairly good for a long time though a

certain degree of anæmia is often present. A few cases waste rapidly. The course of the disease may be prolonged, with periods of great improvement and even apparent cure.

*Bronchospirochaetosis in the Lower Animals*—Mendelson in Siam has recently made the interesting observation of the occurrence of a form of bronchopulmonary spirochaetosis in cats.

**Complications**—Pneumonia and bronchopneumonia have been observed. Rhinitis has also been observed.

The sputum may be examined fresh using the dark ground illumination or may be stained using one of the staining methods such as the Fontana Tribondeau may be employed. The *Spirochaeta bronchialis* is generally present in large numbers while bacteria are very few.

**Differential Diagnosis**—The acute type is often mistaken for influenza or malaria. The examination of the sputum will distinguish the affection from influenza and the examination of the blood will exclude malaria. Cases of the subacute and chronic type presenting blood in the expectoration are generally diagnosed as phthisis. The examination of the sputum for tubercular bacilli will be always negative and the animal inoculations will remain without effect. The ophthalmic and cuti reactions are negative in the great majority of cases. Occasionally however cases of mixed infections of tuberculosis and spirochaetosis occur. From bronchomycosis the affection is distinguished by the absence of fungi cases of double infection, however bronchospirochaetosis and bronchomycosis may at times be observed though very rarely.

Spirochaetosis is easily distinguished from endemic hemoptysis by the examination of the sputum which will show absence of the *Paragonimus ringeri* Cobbold and from bronchomycosis by the absence of fungi.

**Prognosis**—The prognosis is favourable *quoad vitam* but the disease may take a chronic course with anæmia and wasting.

**Treatment**—In the acute cases all the symptoms as a rule disappear after a few days rest in bed. Codeine (½ grain) and pirin (5 grains) may be administered when the cough is painful and the patient complains of rheumatoid pains. In the subacute and chronic types of the disease arsenic introduced by Castellani's treatment of the malarial fever gives good results. It may be administered by the mouth in the form of liquor arsenicalis or arsenious acid pills or may be given subcutaneously in the form of sodylates. Platt and Gill Valerio recommend salarsan when the expectoration is profuse glycerophosphates and bismuth are useful.

In certain cases tartar emetic, especially if combined with arsenic is efficacious. The following formula may be used—Tartar emetic,  $\frac{1}{2}$  grain, liquor arsenicalis 2-3 minims, codein,  $\frac{1}{2}$  grain, glycerine, 1 drachm syrup of

be found  
grains,  
drachm  
day well

diluted with water

Dalmier recommends injections of camphorated oil with gomenol in acute cases and Liquor Fowleri in cinchona wine in chronic cases

### TROPICAL BRONCHOMYCOSES.

**General Remarks.**—Affections of the bronchi and lungs due to

be due to a  
—  
dium Link  
ryptococcus  
rist, 1898  
06  
isan, 1899,

igmatocystis  
heli 1729,

*Rhizomucor* Lucet et Costantin 1900, *Lichtheimia* Vuillemin 1904

5 Due to fungi of the genus *Sporotrichum* Link 1809

For description of above fungi the reader is referred to Chapters XXXVII, p 967 XXXVIII p 978 and XXXIX p 1035

The symp'toms are somewhat similar whichever fungus is the ætiological factor. In *mild cases* there are signs of slight bronchitis with muco purulent expectoration in which the fungi are found. In *severe cases* the patient presents all the symptoms of phthisis with hectic fever and hæmorrhagic expectoration

Mild cases may get cured spontaneously, but they are often benefited by potassium iodide. We will describe in detail the forms of bronchomycosis which has been more completely investigated

#### Bronchomoniasis.

**Synonyms.**—Broncho alveolar moniasis (Castellani) Broncho histomycosis *pro parte*

— of

described several new species of *Monilia* *castellani* recently been confirmed by Pijper, Pantin and other observers. Magrou has recorded a case in France in 1916

**Geographical Distribution**—The disease is found all over the tropics especially in places with a damp climate such as Ceylon and the Malay Peninsula. The affection may be met with also in Europe and America cases having been recently recorded by Pinoy, Iacono and others.

**Etiology**—In Ceylon the malady is generally due to *Monilia tropicalis* Castellani 1910. The same fungus may be found in cases coming from South India and the Malay States. It would appear that the fungus is the real cause of the disease as no other etiologic agents such as the tubercle bacillus, etc. are found for or when the patient gets better the fungus becomes very scanty or disappears completely. In some cases other species of the fungus may be observed but it is doubtful whether all of these

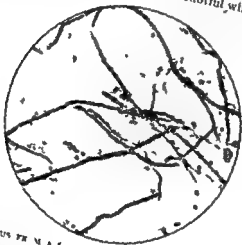


FIG. 771.—FUNGUS FROM A CASE OF BRONCHOMYLIASIS (UNDETERMINED)

are really pathogenic. These species are *Monilia paratropicalis* Castellani, *M. pinoyi* Castellani, *M. guilhermonis* Castellani, *M. nigra* Castellani, *M. cinisida* Castellani, *M. nuda* Castellani, *M. pseudotropicalis* Castellani, *M. lactea* Castellani, *M. frutescens* Castellani, and other monilias among which *M. bethalensis* Papanicolaou 1929. The infection may take place from man to man and also probably from the fungus living saprophytically in nature. *Monilia* like fungi are extremely common in Ceylon in the dust and it is very probable that the so-called "factory cough" is a type of moniliasis.

**Symptomatology**—A mild and a severe type of the malady may be distinguished. In the mild type the general condition of the patient is fairly good there is no fever and he complains



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spontaneously, or, continuing, may turn into the severe type. The severe type closely resembles phthisis, the patient becomes emaciated, there is hectic fever muco-purulent and bloody expectoration. Occasionally true hæmoptysis occurs, a teaspoonful or more of bright blood being spat up at a time. The physical examination of the chest may show patches of dulness fine crepitations and pleural rubbing. This type is often fatal. Between these two extreme types there are of course cases of intermediate severity apyretic or with subcontinuous and continuous fever and more or less marked bronchial and broncho-alveolar symptoms.



FIG 774—SPUTUM IN BRONCHOMYCIASIS (SEVERE CASE)  
(From a preparation stained with methylene blue)

**Diagnosis**—The diagnosis of moniliasis is based on finding the fungus in the sputum. It is absolutely necessary that this should be collected in sterile petri dishes and examined as soon as possible, as sputum exposed to the air becomes contaminated with all sorts of fungi in the tropics. In fresh preparations of the expectoration spore-like roundish or oval cells 4 to 6  $\mu$  are seen often presenting a double contour alone or more rarely with some mycelial articles. The fungus is Gram positive.

the medium

On *gelatins* all the species grow fairly well, a few including *Monilia*

(10 11 12 13 14 15)

**Differential Diagnosis**—Primary bronchomontiasis as described in this chapter should be distinguished from the secondary bronchomontiasis occasionally met with in cachectic patients suffering from cancer diabetes tuberculosis etc. In such cases there is generally thrush of the oral mucosa and the thrush *Moniliae* spread to the pharynx larynx and bronchial mucosa while in primary

spirochetes though occasionally cases of mixed infection occur from endemic hemoptysis by the absence of the ova of *Paragonimus fischeri* Cobbold

**Prognosis**—The cases of a mild type may recover spontaneously or under appropriate treatment. Those of the severe type usually end fatally.

**Treatment**—Mild cases and those of medium gravity respond often to potassium iodide (gr. x to xx) given well diluted in water or milk three times daily. When potassium iodide causes severe symptoms of iodism salicin in the same doses (in cachets) may be administered. In the cases of the severe type we have seen no improvement from the many different treatments administered. Potassium iodide however should always be tried also in these cases as well as bismuths. The diet should be nourishing hypophosphates and glycerophosphates may be tried to keep up the strength of the patient as in phthisis.

#### Broncho-Oidiosis.

16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 32 33 34 35 36 37 38 39 40 41 42 43 44 45 46 47 48 49 50 51 52 53 54 55 56 57 58 59 60 61 62 63 64 65 66 67 68 69 70 71 72 73 74 75 76 77 78 79 80 81 82 83 84 85 86 87 88 89 90 91 92 93 94 95 96 97 98 99 100

**Ætiology** —The following species of the genus *Oidium* Link have been found in cases of bronchitis although it is doubtful whether they are all pathogenic —

*Oidium lactis* Link including *O. lactis* A variety described by Linossier

*Oidium matalense* Castellani

*Oidium rotundatum* Castellani

*Oidium asteroides* Castellani

For description of these fungi the reader is referred to Chapter XXXIX p 1093

**Symptomatology** —This is identical with that of bronchomycosis and two types may be distinguished the mild type and the severe one

**Prognosis** —Favourable in a certain number of cases but cases are met with which do not respond to any treatment and terminate fatally

**Diagnosis** —This can be made only by cultural methods

**Treatment** —Potassium iodide should be tried in all cases but

### ||Bronchohemisporosis

**General Remarks** —This bronchomycosis has been described by Castellani The fungus found so far is *Hemispora rigosa* Castellani a description of which will be found in Chapter XXXIX p 1108

patches of dulness fine crepitations pleural rubbing  
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**Complications** —The affection may be complicated with tonsillitis caused by the same fungus and characterized by the presence of yellowish or greyish patches at other times the

### Tea Factory Cough

This affection described by Castellani in 1910 is common in Ceylon It is probably a form of broncho mycosis Coolies

working in tea factories are occasionally observed to deteriorate

occasionally a few coarse râles. If these coolies are taken away from the factory and sent to work in the fields, all the symptoms slowly disappear.

A similar affection may be observed in tea-tasters. Tea-tasters, in order to judge of the quality of teas, not only taste infusions but frequently fill their hands with the leaves and bury their noses in them, snuffing them up, in this way a certain amount of tea dust enters their nasal cavities and with the tea-dust the micro-organisms which are found in it. According to the researches of

*mal'oculis*, and *Pent-*

4. A peculiar *streptococcus*, somewhat different from *S. pyogenes*, frequently

It is interesting to note that such germs are very rare or absent in samples of tea examined in England. The same organisms may be found in Ceylon in nasal cavities of tea-tasters, in their expectoration the *Monilia* like fungi are practically constant, the *streptococci* very frequent while *Aspergillus* and *Penicillium* fungi are rare. Guinea pigs in whose nostrils tea dust is daily insufflated for months develop a bronchio alveolar moniliasis.

#### Bronchial Aspergillosis.

It is probable that the deleterious effects caused by the fungi are due not only to a mechanical irritative action but also to toxins secreted by the fungi.

**Morbid Anatomy.**—The lungs and occasionally the liver, kidneys and other organs may show a type of pseudo-tuberculosis characterized by the presence of numerous *tufts* subserous like nodules.

**Symptomatology.**—The presence of the fungi in the bronchi when in small amount, may not give rise to any symptoms. When the infection is heavy,

dy expect  
ve hectic

nd that n  
ations a e

therefore necessary

**Prognosis**—Mild cases often recover but in severe cases with bloody ex

n the condition is brought about  
on is very beneficial Potassium

### Bronchial Penicilliosis

### Bronchial Mucormycosis

is in the genus *Mucor* Michx. 1729  
Dk. 1729

fung see p 972

### Bronchial Sporotrichosis

A few cases have been described of a bronchitis due to fungus of the genus  
*Sporotrichum* Link 809

### Undetermined Bronchomycosis

One of us has described cases of bronchomycosis due to fungus which have not  
yet been classified

## PULMONARY NOCARDIASIS

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ance somewhat resembling the tubercle bacillus. Other species of nocardia, also found in sputum may not be acid fast, and these are more easily recognized.

**Morbid Anatomy.**—As a rule the appearances found post mortem are not unlike that of tuberculosis. There is consolidation, necrosis and cavity formation in the lung with or without the signs of caseous pneumonia or of calcareous deposition or there may be small curvilinear nodules scattered through the lung. There may also be nodules in the liver spleen peritoneum and lymph glands and there may be chocolate-coloured exudate into the pleural or peritoneal cavities. This exudate may be odourless or foetid in colour. The fungus can be easily found in these pseudo tubercles.

**Symptomatology.**—In general it may be stated that the symptoms resemble those of phthisis and as such the history is usually recognized. Usually there is fever, cough and sputum containing blood at times and showing acid fast rods resembling the *Bacillus tuberculosis* but careful search may reveal a few elongate or branching forms. The physical signs are those of chronic broncho pneumonia with or without cavity formation and with or without those of pleural effusion. The liver and spleen are often enlarged. The cases usually go from bad to worse and end in death.

**Diagnosis.**—Many of these cases are diagnosed as pulmonary tuberculosis at the present time. The correct diagnosis can only be established by a careful examination of the sputum by microscopical and bacteriological methods including the culture of the organism.

The differential diagnosis has to be made from phthisis and liver abscess. It may be distinguished from phthisis by the recognition of the beaded, branched organisms in the sputum and the culture of it therefrom. In cases giving a history of dysentery and exhibiting enlargement of the liver, fever and a purulent chocolate-coloured effusion into the pleural cavity the diagnosis can only be effected by finding the fungus and by the absence of any pus in the liver. In such cases the dysentery amoeba may be present in the faeces.

For the morphological and cultural characters of the species of nocardia see Chapter XXXIX p 1010.

**Prognosis.**—So far the prognosis is very bad as all known cases have died.

**Treatment.**—Iodide of potash in large doses may be tried or a vaccine made from the patient's causal organism.

**Prophylaxis.**—Nothing whatever can be said on this part of the subject.

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## CHAPTER LXXII

# DISEASES OF THE CIRCULATORY SYSTEM

General remarks—The blood—Blood puzzles—Anæmia and allied conditions—  
Leukæmia—General dropsy—The heart—The vessels—The spleen—  
The bone-marrow—References

### GENERAL REMARKS

In this chapter we merely make a few allusions to some points of tropical importance with regard to the blood and the organs which produce and circulate it. The subject is not merely a large one but is of great importance in the tropics and our few remarks are merely of an introductory nature.

### THE BLOOD.

We in no way intend to enter at all fully into an important study of the blood which is to be found in detail in the special books devoted to its elucidation but we desire in the briefest manner possible to present to the reader a few remarks which have a direct bearing upon the various references which we have made from time to time in the preceding chapters with regard to it.

#### The Erythrocyte.

In embryonic life the first sign of the blood cell is to be found in those mesoblast cells of the vascular area which contain hæmoglobin

marrow of post embryonic existence

These normoblasts multiplying by mitosis are the source of the erythrocyte which alone is the proper denizen of the circulating blood.



which lies the *stroma* in the form of a network of protoplasmic threads and nodes which contains the hæmoglobin and in the saucer shaped corpuscle forms a cap over the archoplasm which lies just over the concavity of the saucer

Excentrically placed and surrounded by the glassy body lies the *capsule corpuscle* near which lie two sharply defined bodies embedded in a substance which may contain a vacuole These are the *centrioles*

3 The *blood plate* is the metamorphosed nucleus of the normoblast which lies excentrically on one side of the archoplasm It is easily extruded in making the blood film and gives rise to the blood platelets which are so well known Its peculiar appearance is possibly due to physiological modification during mitosis of the normoblast

4 *Mies plastokonten* are granular bodies of unknown nature scattered through the erythrocyte but mostly seen near the archoplasm

As we shall see later these various parts of the erythrocyte are the explanation of the intracorpuseular blood puzzles and the origin of the numerous intracorpuseular pseudo parasites of many observers

The red corpuscles number some five millions in a healthy male and some four and a half millions in a healthy adult female under the climacteric age but there are physiological increases of these numbers in infancy by cold and at high altitudes while race has but little influence

Pathologically they can be increased by mechanical means—e.g. the concentration of the blood caused by diarrhoea sweating vomiting and polyuria or by heart disease They can also be increased in toxic conditions and in polycythemic splenomegaly They may be decreased by any mechanical toxic or parasitic cause which induces blood destruction

The normoblasts of the bone marrow may accidentally occur in the normal circulating blood as an isolated form but in numbers they indicate that there is an abnormal demand for erythrocytes—as for example after a hæmorrhage A normoblast is about the same size as an erythrocyte (7.5 microns) but contains a rounded nucleus composed of a nuclear membrane containing dense chro-

ome  
size  
large

nucleus measuring some 10 microns and containing loosely arranged chromatin The nucleus may undergo the usual changes and the megaloblast become a megalocyte

Microblasts are small cells or abnormal normoblasts or megaloblasts

## The Leucocyte

In post-embryonic life the home of the leucocyte or colourless blood cell is the bone marrow. They may be divided into the hyaline cells or lymphocytes which are the least differentiated cells both ontogenetically and phylogenetically, being the first

belonging to the same denomination. According to Heidenhain there is a tendency on the part of the centrosome to reach the centre of the lymphocyte being pulled thereto by cytoplasmic radii. It is however prevented from assuming this position by the nucleus which succeeds in this obstruction in the case of the small and

somewhat smaller it possesses a rounded or slightly indented nucleus colouring deeply with basic stains and surrounded by a very slight amount of cytoplasm which shows a reticulum with basophilic granules as the nodal points. There are also some azurophilic granules.

The large lymphocyte is larger reaching to 20 microns and is characterized by a central roundish or slightly indented nucleus. There is  
ly but

The large mononuclear leucocyte possesses an excentric nucleus relatively poor in chromatin surrounded by a larger amount of cytoplasm with the usual reticulum and azurophilic granules.

The transitional cell has its nucleus indented into a horseshoe or sometimes twisted. Lymphocytes and myeloblasts may contain suchsinophile granules called Schridde's granules. The true leucocytes or granular white cells are classified according to their granules into neutrophile, eosinophil and basophil. Their parents are the myelocytes of the bone-marrow which are cells of large size

The polymorphonuclear neutrophile leucocyte is generally called by the first portion of its name. It varies considerably in size and is characterized by a lobulated nucleus in which the lobules vary in number but however separate they may appear are always connected by fine threads. It is upon this lobulation that the *Arneth count* is based and the form of the nucleus can be explained in the same way as in the transitional (*vide supra*)

The basophile leucocyte or mast cell has a trilobed nucleus poor in chromatin and a cytoplasm containing basophile granules. The

They are triangular with an excentric nucleus and a markedly basophile granular cytoplasm.

The number of white cells varies from time to time from about 5 000 to 9 000 per cubic millimetre but the average is about 7 000 or one to every 700 erythrocytes.

Following Gulland and Goodall the average differential count is as follows —

Polymorphonuclear neutrophile leucocytes	70	} Average
Small lymphocytes	20	
Large lymphocytes	5	} Maximum
Eosinophile leucocytes	4	
Basophile leucocytes	1	

The *Arneth count* formulated in 1904 is based upon the number of lobules in the nucleus of 100 polymorphonuclear or 100 eosinophile leucocytes. Class I contains those with a single rounded or in

added together give the Arneth index and in Classes I, II and half III give the Bushnell-Trenholtz index. Arneth subdivided each class into smaller groups by the indentations and the character of the loops and lobes but these are not now considered. He restricted his counts to polymorphonuclears but to-day eosinophile leucocytes are also considered. In counting it is wise to follow Chamberlain and Vedder and to consider as one lobules with a

It is evidence of lower resistance on the part of the patient to a disease. This count has been tested in the tropics by Chamberlain and Vedder Macfie Breinl and others as has been set forth on p. 75.

A few examples may be given as follows —

### ARNETH COUNT

Kind of Leucocyte	Nature of Observation	Observer	Class I	Class II	Class III	Class IV	Class V	Arneith Index
Polymorphs	Normal in Europe	Arneith	5	33	41	17	240	400
Polymorphs	Normal in America	Hagan	5	19	41	25	54	240
Polymorphs	Normal Americans in Philippines	Chamberlain and Vedder	13.3	32.3	37.2	14.6	20	46.2
Polymorphs	Normal Philippines	Chamberlain and Vedder	27.3	35.1	25.8	7.5	0.9	63.8
Polymorphs	Normal limits	Simon	4.9	21.47	33.44	9.23	2.4	25.56
Leucocytes	Normal in Europe	Arneith	11	69	19	1	—	80

Von Schilling Torau suggests a modification which is to classify the neutrophils as myelocytes myelocytes with indented nucleus polymorphs with rod shaped nuclei polymorphs with segmented nuclei. It is said that this simple method gives the same results as the Arneith count.

The Arneith count is being used at present in tropical work but its value is still *sub judice*.

The leucocytes vary in number under physiological and pathological conditions —

Frequently in ...

fever tuberculosis an influenza or in excretoriums of any kind. The leucopenia is usually polymorph nuclear but in fevers there is a diminution of eosinophiles.

**Leucocytosis**, or increase in their numbers may occur in physiological conditions such as infancy, pregnancy digestion and exercise and there is a terminal or agonal leucocytosis before death. In pathological conditions a leucocytosis due to polymorphonuclear leucocytes may occur in septic or inflammatory conditions many fevers and toxæmias after hæmorrhage and with malignant disease.

**Lymphocytosis** may be relative when there is a relatively high percentage of lymphocytes and low of polymorphs with no increase in the total number of white cells or absolute when the total number of white cells as well as of lymphocytes is increased. The former occurs in protozoal infections such as malaria amœbic dysentery etc and the latter in leukæmia etc.

**Eosinophilia**, or increase in the eosinophile leucocytes occurs in helminth infections in skin diseases in asthma in toxic states and in myelocythæmia.

**Basophilia** occurs in myelocythæmia and staphylococcal infections but in the latter only slightly.

### Blood Platelets

These are probably derived from the erythrocytes and may

health. They are colourless refractile discoidal bodies some 1-3 microns in diameter having a great tendency to adhere together and having an affinity for basic dyes.

### Hæmoconia

These are colourless refractile bodies 0.5-4 microns in diameter which do not colour with ordinary stains and are of unknown origin and function though they may be fat particles as shown by Neumann.

### BLOOD PUZZLES

Blood puzzles consist of bodies which from the first have been recognized as such or in other instances have been thought to be parasites.

It is difficult to give a systematic account of these bodies but following Balfour we may classify them as follows —

- A Heterogenetic — Not in the blood
- B Autogenetic — Actually in the blood
  - I Found in fresh blood —
    - (a) Erythrocytic
    - (b) Leucocytic
  - II Found in stained blood —
    - (a) Erythrocytic
    - (b) Leucocytic or lymphocytic

## Heterogenetic.

These are external or adventitious and may be divided into —

- I Those belonging to the glass slide
- II "
- III "
- IV "
- V "

**The Glass Slide**—Everyone is well aware of the peculiar marks which may appear on old glass slides and which retain the stain thus giving rise to pseudo-trypinosomes yeasts and many other forms.

Perhaps the most interesting of these are the 'X bodies' (Horrocks and Howell) which appear in Romanowsky films as reddish bodies with a small blue circular centre surrounded by four or more faint concentrically arranged capsules and which Chamberlain and Vedder have shown to be artefacts present in the glass slide.

**Cleaning and Drying Materials**—Cotton fibres may be introduced from a cloth in cleaning slides. Blotting paper if used twice for drying blood slides may introduce one kind of blood into another or blot it into a blood film.

**The Air**—Insect scales, plant hairs, animal hairs, yeasts and

**The Intestine**—In obtaining films during a post mortem or from an animal which has been shot there is danger of contamination of the blood with spirochetes and other organisms from the intestine.

## Autogenetic

These are bodies which are really in the blood whether natural products or artificial productions.

**Fresh Blood**—**ERYTHROCYTE**—In anemic blood the erythrocytes

A crenation seen in a deformed or in an ordinary corpuscle may in certain focal places look like a malarial parasite.

Vacuols have clear cut margins do not move do not possess filament and are quite clear yet they give rise to tremor and may be mistaken for proplasma or for malarial parasites.

The glassy body crenation above when closely visible is often mistaken for a parasite and is probably the explanation of the malarianous or delirious hazy spots which have been de-

scribed as well as of such pseudo-parasites as those mentioned by Balfour as being described by Foran and Breeze. The dark spot mentioned as being visible in the malarial plasmodium may be the centrosome.

A granule from a leucocyte or a blood platelet lying on an erythrocyte may simulate a parasite.

**LEUCOCYTE**—The leucocyte especially the eosinophile is responsible for the free granules and worse for the free or attached wavy process which is apt to be mistaken for a spirochæte especially when a beginner is using the dark ground illumination.

**Stained Blood**—**ERYTHROCYTE**—When the glassy body swells it gives rise to pale large red blood cells 15-50 microns in diameter which are the half moon shaped or sickle shaped corpuscles of Stephens and Christophers.

The hæmolyzed stroma is the cause of the shadow corpuscles and polychromatophilia is due to diffuse colouring of the reticulum while the punctate form is due to the nodes being especially tinted.

Schüffner's dots are caused by the coloration of nodes of the reticulum in older cells while pathological karyolysis of the nuclear plate may be the cause of the Howell Jolly bodies and the ring shaped bodies of Cabot and perhaps the so-called *Paraplasma flavigenum* is due to the same cause.

The capsule corpuscle may be the origin of Arnold's nucleoids, Schmauch's bodies, Heinz's corpuscles and many pseudo parasites.

A blood platelet lying on an erythrocyte may resemble a malarial parasite.

**LEUCOCYTE**—The puzzles in connection with the leucocyte or lymphocyte may be divided into—

- 1 Extranuclear
- 2 Intranuclear

**Extranuclear**—Kurloff's bodies seen in the large lymphocytes especially in guinea structure and may parasites. They stain

constantly show dots of chromatin and measure 2-6 microns in diameter. They also occur free in the liquor sanguinis and are by him

## BLOOD PARASITES.

The blood may contain a number of parasites—e.g. the malarial parasites the spirochetes the trypanosomes the kala-azar parasites—as well as certain worms—e.g. the *Microfilarie Schistosoma hematobium S japonicum* and *S mansoni*

## ANÆMIA AND ALLIED CONDITIONS

Anæmia is common being generally associated with ankylostomiasis malaria kala-azar chronic dysentery or in women repeated pregnancies and prolonged lactation. It is especially common among coolies working on estates. The treatment is to remove the causal agent and then to administer the old mixture of sulphate of iron sulphate of magnesium and nux vomica to the poorer classes while intramuscular injections of iron alone or combined with arsenic or sodium glycerophosphate are more scientific and more suitable for the better classes.

Chlorosis is rare but we have seen cases in Europeans and in better class native girls.

## Paranæmia Tropicalis.

Everyone residing in the tropics is acquainted with the pallor which is visible in the faces of many European residents who apparently are in good health. An examination of the blood fails to reveal any marked diminution in the red cells or hæmoglobin or at all events no such reduction as would be compatible with the pallor. We have used the term paranæmia to indicate the condition.

In Chapter III, section Effects on the Blood (p. 75) we have shown that this apparent anæmia has been carefully studied by W. M. Strong who considers that this pallor is really caused by the deposition of pigment in the epidermis. This pigment renders the skin opaque to the red rays contained in sunlight and hence the colour reflected therefrom appears to the eye white.

In diagnosing this condition care must be taken to exclude true anæmia by a count of the red cells in the blood and an estimation of the hæmoglobin.

## LEUKÆMIA.

Leucocythæmia cannot be said to be very rare at all events in our experience and may be either spleno-medullary or lymphatic in type. It is most necessary to remember the possibility of the occurrence of this disease and to make it a rule to examine the blood microscopically before performing splenic or hepatic puncture in cases of splenomegaly.

Pseudo-leukæmia and Banti's disease are also known



### GENERAL DROPSY

Cases of general dropsy not due to heart or renal disease may be caused by beri beri or ankylostomiasis

### THE HEART

Heart disease has not been carefully studied by modern methods in the tropics but cases of heart block due to malaria and syphilis have been recorded by us

pro  
to  
heart especially brown atrophy are quite common as the result of some general disease Heart block is rare but has been met with Rupture of a perfectly normal myocardium the pericardium being intact is recorded by Herzog as due to fracture of the second fourth and fifth ribs

#### Tropical Heart

Under this heading MacLeod has described the conditions of palpitation and dyspnoea on going up hills met with in persons who

believes that the condition is associated with subnormal blood coagulability and recommends the administration of calcium salts The salt he prefers is calcium lactate which he gives in 10 grain

citric acid or its salts should be avoided when calcium salts are administered as they increase the calcium excretion

### THE VESSELS

*Atheromatous* degeneration of the arteries is quite common and aneurysm is found generally affecting the thoracic aorta while varicose veins and varicocele are usual and associated with hypertrophy of the heart are very common among rickshaw coolies

*Thrombosis* is often met with as the result of typhoid fever and other diseases and we have seen thrombosis of a coronary artery with myomalacia cordis or aneurysm of the heart

### THE SPLEEN

The spleen is affected in malaria kala azar relapsing fever etc as already described Capsulitis is very commonly met with in

post-mortems, but splenic abscess is, in our experience, rare, and may be of entamœbic origin. Infarcts and tuberculosis are, however, not so rare, and spleno medullary leukaemia, as has been mentioned above is not very rare. Rupture of the enlarged malarial spleen has already been mentioned and may cause death within a few minutes or the patient may live for several hours.

### BOVE-MARROW.

The importance of the bone marrow is often overlooked in the tropics. It requires especial study in anamias kala azar, and malaria.

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## CHAPTER LXXXIII

# DISEASES OF THE LIVER AND PANCREAS

General remarks—Tropical liver—Amoebic abscess of the liver—  
Opisthorchiasis—Clonorchiasis—References

### GENERAL REMARKS.

DISEASES of the liver and pancreas are of common occurrence in the tropics. *The liver* may be affected in the course of tropical fevers especially in malaria and kala azar, in the latter of which Rogers has described a special form of cirrhosis. The disease called '*infantile biliary cirrhosis of the liver*' described by Ghose and Mackenzie as occurring in Calcutta and other parts of India in Hindu and Mohammedan children appears to us to require reinvestigation with a view to deciding whether it also is a variety of kala azar. It is said to attack children under one year of age and to be characterized by a low type of fever, associated with enlargement of the liver and spleen jaundice, pale motions dark urine, and sometimes vomiting of blood œdema and ascites and ends fatally in three to eight months.

*Acute yellow atrophy* of the liver is not as uncommon in Ceylon as in Europe for on an average we have met with one or two cases per annum. It occurs in Ceylon more commonly in men than in women but the cause appears to be quite obscure.

An extraordinary case of acute severe hepatitis and gastritis which caused a considerable hæmorrhage to take place filling all the small biliary ducts the gall bladder the common bile-duct the duodenum jejunum and ileum with blood has been recorded by one of us in Ceylon. The inflammation occurred in a stomach

GENERAL REMARKS

reguent than is admitted by many authors. It will be shown presently that various parasites cause cirrhosis.

Primary cancer of the liver has been met with but is very rare. On the other hand secondary cancer is by no means rare. The only non malignant growth which we have met with was an angioma.

The protozoal parasites which occur in the liver are *Loeschia histolytica* the cause of liver abscess. *Lishmania donovani* and the malarial parasites as has already been noted. Coccidiosis has been found in the tropics in man several times.

With regard to the trematode parasites of the liver *Ascarola hepatica* is only an occasional parasite of man. *Ascarolopsis hepatica* has been seen in a Chinese who showed obscure liver symptoms and no doubt microscopic examination of the faeces would make diagnosis possible but there is only one certain case, which will be considered later as will clonorchiosis caused by *Clonorchis sinensis*. *Dicrocoelium lanceolatum* is considered to be the most likely to cause any serious symptoms while living in the bile ducts. *Schistosoma mansoni* and *S. japonicum* may both affect the liver (pp 157) 1867

With regard to tape worms *Taenia chinensis* is not common but we have met with one case of echinococcus in Ceylon which was brought by a Moor prisoner and Begbir has recorded another in the liver but associated with the lung in an old resident in Ceylon. One of us has recorded in invasion of the liver in Swaziland and the formation of abscesses by the agency of the worms. We have also recorded *Paraccephalus armulatus* in liver.

Disease of the gall bladder is common in the tropics and we meet with a wide and characteristic inflammation (gall stones are met with probably as sequelae to typhoid fever but also as a result of other causes. Suppuration of the bile ducts we have seen once in India. Stricture and catarrhal jaundice are met only so infrequently in the tropics.

A cyst of the liver may take place as the result of trauma and recently Herzog has recorded this accident in a child in a fracture on a cloth wound round the abdomen. In this rupture had been caused by the perforation of the tip of the rib into the left liver.

The pancreas in our experience is not infrequently diseased. We have met with the following types of it

- 3 Subacute pancreatitis
- 4 Chronic pancreatitis
  - (1) Syphilitic in the foetus
  - (2) In the adult
    - (a) Chronic interlobular pancreatitis
    - (b) Chronic interacinar pancreatitis
    - (c) Chronic interlobular and interacinar pancreatitis combined

It should be remembered that the subtertian parasite is one of the causes of *hæmorrhagic* pancreatitis. In addition we have seen cancer of the pancreas producing a blocking of the duct of Wirsung and leading to a ranula which contained many pancreatic calculi. We have once seen an ascaris in the duct of Wirsung associated

liver congestion  
monorchosis

### TROPICAL LIVER

**Synonyms**—Congestion of the liver Hyperæmia of the liver  
Indian liver

**Definition**—Congestion of the liver is a hyperæmia brought about by many conditions especially gastro intestinal disorders

**Remarks**—There can be no doubt that the European is apt to eat and drink more than is good for him on his first arrival in the tropics and that this is bound to lead to an increase of blood in the liver, which physiological condition may easily become a congestion with later blood stasis and diminution of the functional activity of the organ

**Climatology**.—Congestion of the liver is a cosmopolitan complaint but is much more commonly met with in the tropics than

tary canal as well as those which have laboured with  
patient

**Symptomatology**—The illness begins with frontal headache  
tongue is  
tated with  
The liver  
light in the  
or in the

## TROPICAL LIVER

to shoulder and there may be slight signs of jaundice in the low conjunctiva and sallow skin. The urine may be diminished in quantity, high-coloured and with high specific gravity and loaded with uric acid and urates. The nervous system is also affected and the patient is usually very cross and irritable. The temperature is generally normal but sometimes it rises and when above  $100^{\circ} \text{F}$  it is usual to call the disease hepatitis instead of congestion of the liver. The common non febrile variety lasts from two to seven days but is liable to recur.

**Varieties**—Congestion of the liver may be acute when associated with some other disease or chronic when due to gastro-intestinal disturbance.

**Diagnosis**—The diagnosis is based on the painful enlargement of the liver which in the absence of other disease is usually unaccompanied with marked rise of temperature.

**Treatment**—In the acute attack it is as well to keep the patient in bed and begin the treatment with a dose of colamel (gr ii to gr v) followed a few hours later by a saline in the form of magnesium and sodium sulphate or Carlsbad salts. An effervescent mixture of ammonium carbonate (gr ii to gr v) and sodium bicarbonate (gr xx) with citric acid (gr xv) may be given every three hours or a mixture containing ammonium chloride in some combination. At the same time hot fomentations or a thick coat of antiphlogistic may be applied to the region of the liver. The diet should consist of soups and milk diluted with Vichy or bary water and no alcohol in any form allowed.

When the condition has become chronic the patient must be carefully dieted and placed upon a course of treatment with the effervescent mixture and Vichy (Grande Grille) and when leave in Europe is available should be sent to Vichy Carlsbad Hurreau or Montecatini. He should avoid alcoholic stimulants and rich food of every description and especially tinned food and should restrict his diet to fish, clear soups, fish and well-cooked vegetables and milk avoiding meat and curries.

Whenever the acute attack is over the patient should be advised to take exercise daily, walking, riding, golf or tennis combined with the usual so-called liver exercises.

**Prophylaxis**—Plain simple not highly spiced food should be taken and such pernicious drinks as the heavy forms of beer, champagne etc should be avoided. If any form of alcohol is to be taken in the tropics this should be Scotch whisky well diluted or light clarets and then only in moderation. Chills must be avoided as carefully as possible especially when there is a hard winter in the runs and when changing from a warm to a cooler climate. With regard to the cold bath there is no doubt that the majority of people are unable to stand this in the tropics and therefore some persons are however distinctly benefited by a cold bath. Individual peculiarities must be considered.

The most dan  
as well to sleep  
and in flannel ni  
cholera belt ma  
whenever they become damp

### AMŒBIC ABSCESS OF THE LIVER

Synonyms —Hepatic abscess . . . *French* Absces du Foie . . . *Italian* Fegate Suppurativa . . . *German* Tropischer Leberabszess

Definition —Amœbic abscess of the liver is a suppurative hepatitis caused by *Loeschia histolytica*—usually preceded by an attack of amœbic dysentery

History —Liver abscess was known to the ancients, and was

doctors and the Indian army surgeons. The discovery of *Amœba* or *Loeschia* emphasized the connection between liver abscess and dysentery. Kruse and Pasquale were the first to regularly find amœbæ in the liver abscess and to state that apart from the amœbæ the pus was sterile. Later researches have confirmed this discovery and have demonstrated that the pus of a liver abscess does not contain

and perhaps other varieties of *Loeschia*. It is more common in Europeans than in natives and more so in males than females and is usually a disease of adults. The most important predisposing cause is perhaps alcohol.

In monkeys one of us has recorded the occurrence of liver abscess due to an amœba (*Loeschia* + *Itali* Castellan 1907).

Pathology —The *Loeschia* pass from the bowel via the portal vein into the liver where they produce coagulative necrosis of the liver cells which become formless and break up into granular debris. This necrosis is thought to be brought about by means of toxins produced by the *Loeschia*. The necrosed area undergoes liquefaction and forms the abscess the contents of which consist of

from the local

in the body  
 liver tissue for some distance  
 cess has been opened amœbe  
 T) = process of repair has not been fully  
 formed

to repair

Usually there is only one  
 abscess but it is not uncommon  
 to find two and there may be  
 more. The abscess is generally  
 found in the posterior part of  
 the upper portion of the right  
 lobe. It is rounded in form  
 with walls composed of degener-  
 ated liver cells and granulation  
 tissue. Its contents may be  
 thick, creamy pus but more  
 usually it is yellowish or brown  
 colored. On microscopical ex-  
 amination it consists largely of  
 detritus with a few degenerated  
 liver and pus cells. The bacteria  
 found in the pus when it is not  
 sterile are streptococci, staphy-  
 lococci, *B. coli communis* and  
*B. pyovanicus* and occasionally  
 some anaerobic germs. The  
 abscess varies much in size from  
 a small hollow containing only  
 1 or 2 ounces up to a huge cavity  
 with a couple of pints or more



FIG. 775.—AMOEBIĆ ABSCESS OF THE LIVER

of pus while even larger have been described. The size of the  
 liver apart from the abscess also varies being sometimes increased  
 and sometimes diminished. Apart from the liver abscess there are  
 usually signs of old or recent dysentery in the colon though these  
 may be absent. There may be abscesses in other parts of the body

previous attack  
 disease begins in  
 This is fever  
 sometimes

diminished in the tissue

capitulum is  
 said to be called to



the arched 'cupola like' curve of the upper aspect of the liver, as seen by radioscopy  
 purative stage 'is of  
 nized and appropri  
 stopped in a certain  
 done rigors may take place, and the fever usually becomes more severe, and a typical hectic temperature with night-sweats may ensue while the patient complains of a dragging sensation on the right side pain under the right shoulder blade and in the right shoulder This latter is a referred pain due to the fact that the phrenic nerve arises from the fourth cervical nerve-root, the fibres of which supply the skin of the shoulder

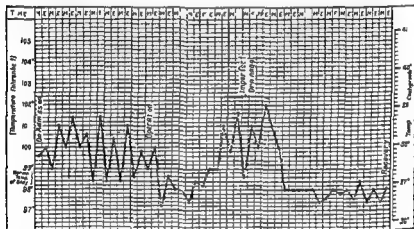


FIG 776 — THE TEMPERATURE CHART OF A CASE OF ABSCESS OF THE LIVER

A patient with an abscess of the liver often possesses a most typical facies. He lies on his back with his legs drawn up, his face is drawn and of a pale yellow colour and the ocular conjunctiva  
 times  
 tant  
 pain  
 ful, hence the breathing is costal in character and often there is a slight cough. The hands and feet are often cold and clammy to the touch.

On the abdomen and chest the right hypochondrium

part of the chest fine crepitations or friction sounds may be heard

78.37 per cent lymphocytes 17.44 per cent mononuclears 3.15 per cent and eosinophiles 0.70 per cent which closely agrees with Rogers figures which are Polymorphonuclears 74 to 87 per cent lymphocytes 7 to 22 per cent mononuclears 3 to 7 per cent, eosinophiles 3 to 4 per cent. In some of our cases there was no

into the lung causing signs of pleurisy and pneumonia associated with the expectoration of characteristic brown or reddish brown gummy viscid purulent matter or into the stomach when a similar material will be vomited or into the bowel when it will be passed *per anum*. It may also burst into the pericardium or the peritonum or into the vena cava all of which cases are bound to end fatally. If it does not burst the patient may die of exhaustion.

The duration of a liver abscess is very variable being from a few weeks to several months and even years.

Diagnosis.—The diagnostic points in abscess of the liver are A history of dysentery fever generally of a serotinic type with

the characteristic

rectus the low

and the frequent

above all the discovery of the pus by exploratory puncture as described below

The differential diagnosis between the pre-suppurative and the suppurative stages is often impossible without a puncture but sweating high intermittent temperature if present and not influenced by the emetine treatment is suspicious that suppurative

the dullness is horizontal while in liver abscess it is convex

side the case is probably one of liver abscess. It is to be noted, however, that a right pleural effusion and a liver abscess may

enlarged spleen. In those cases of liver abscess in which fever is the only symptom the diagnosis may be extremely difficult and may require all modern bacteriological methods to exclude Malta

cholecystitis in most cases gives no trouble on diagnosis as the enlarged gall bladder can be easily felt and there is generally a history of hepatic colic

Syphilitic gumma of the liver may show many symptoms in common with liver abscess including the intermittent fever as we have twice noted. The positive Wasserman reaction and the action of potassium iodide in full doses clears the diagnosis

Cases of leukæmia pseudo-leukæmia tropical splenomegaly and kala azar have been mistaken for liver abscess but in all these conditions the spleen is also greatly enlarged

In this connection we may emphasize the necessity for the examination of the blood in order to exclude leukæmia otherwise a fatal hæmorrhage may follow such a simple operation as puncture of the liver. Liver abscess is usually easily diagnosed from hydatid disease but when purulent changes have taken place in the latter the diagnosis may be impossible except by the history and the eosinophilia

**Prognosis**—If the abscess is left unoperated for a long time the prognosis is very poor. If the abscess is operated on the prognosis is also bad but

operative measures have come into more common use the mortality has decreased according to Dujardin Beaumetz from 82 per cent to 32 per cent. The operation wound may occasionally become phagedænic

**Treatment**—If a case is suspected by the symptoms and blood examination, the patient should be treated with emetine or ipecacuanha. The precautions to be taken are the same as for typhoid fever. The latter must be continued for several weeks after every sign of hepatitis has disappeared

If, however, an abscess is believed to have formed, there should

once if necessary.

The needle should be driven into the liver in the region of any definite swelling or pain or fading these through the eighth intercostal space in the anterior axillary line about 1 or  $1\frac{1}{2}$  inches from the costal margin.

The direction of the needle should be inward slightly upward and backward because the usual site of an abscess is in the upper and back part of the right lobe.

Aspiration by the syringe or the aspirator may reveal pus or may fail to do so, in which latter event the needle must be carefully and slowly withdrawn and its contents ejected on to a clean white dish to see if it is composed of the grumous material of liver abscess. The needle should now be driven into the liver in different places

to determine hepatic phlebotomy. When the abscess has been located the needle should be left *in situ* as a guide and one of two procedures may be carried out: A. Aspiration B. Operation.

The operation should however always be performed if the abscess has already burst into the lungs, the peritoneal or pleural cavities.

**A. ASPIRATION.**—This is preferable if the abscess is small and consists of evacuating its contents and the injection of a solution of 1 grain of emetin hydrobromide in 2 ounces of water, or the repeated irrigation of the cavity with a solution of bihydrochloride of junine (3 to 5 grains to the ounce) by means of Royle's flexible

NET INCISIONS

The site of the operation depends upon where the pus has been situated. Two principal places may be mentioned: (1) through

the evacuation of the pus. This may be useful in certain cases when there are no signs indicating the position of the abscess.

**2. Operation Through the Thoracic Wall.**—An incision should be made through the parietes including the site of the puncture.

through which pus was obtained. After the skin has been well retracted a piece of a rib may require to be removed thus exposing the diaphragm below the pleura which must be incised and stitched to the margins of the wound and the wound well packed with gauze.

The liver is now exposed and two methods of procedure are open to the operator—either to push a pair of dilating forceps along the needle which has been left *in situ* and thus to open up the cavity and evacuate the pus and then after inserting a double drainage tube to wash out the cavity with the quinine solution mentioned

Manson's apparatus can of course be used in this position as well as through the thoracic wall.

**Post-Operative Treatment**—The dangers of the operation itself are but slight. The pleura may be opened and if this happens it should be carefully closed by stitches.

The post operative complications are many and include hæmorrhage

this does not happen ■ second abscess or one of the above complications may be suspected unless indeed it is due to imperfect drainage which must at once be rectified.

taken that the drainage tube is not too rapidly shortened other

possible be given ■ holiday  
 use of emetine or ipecacuanha  
 after the operation wound has quite healed is distinctly indicated  
 in order to prevent the formation of another abscess

## OPISTHORCHIOSIS.

**Definition.**—Opisthorchiasis is an invasion of the bile-ducts with *Amphimerus nocerca* Braun, 1903 (p 577), and *O felinus* Rivolta, 1885 (p 576)

**Remarks.**—*A nocerca* is said to be common in dogs in India but has only once been found in the dilated bile-ducts of an Indian in Calcutta who died with dysenteric symptoms.

*O felinus* has been found in the dilated bile ducts and incidentally also in the intestine of human beings cats and dogs in Tomsk. It caused a form of hepatic cirrhosis

## CLONORCHIOSIS

**Definition.**—Clonorchiasis is the invasion of the bile-ducts with *Clonorchis sinensis* Cobbold.

**Climatology.**—This parasite occurs in China Indo-China and Japan

**Pathology.**—These parasites occur in dogs and cats but the method of infection is quite unknown. In man they live in the bile-ducts in the recesses in the wall of the dilated gall bladder or of the bile-ducts and in the duodenum. They may also be found in abscesses apparently unconnected with the liver. The ova are found in the alimentary canal and the feces. The liver becomes enlarged and may be darker in colour while it is atrophied in the neighbourhood of the dilated ducts. The intestine may show catarrhal inflammation. Often the spleen is also enlarged and there may be ascites or oedema. Ova have been met with in the abdominal lymphatics while the flukes themselves have been found in a lumbar abscess. The blood shows an eosinophilia.

**Symptomatology.** There is an abnormal appetite but the general health remains good for some time, though the liver enlarges and



FIG. 777 — *Opisthorchis felinus* RIVOLTA  
(After Looss)

**Diagnosis.**—The presence of the worms can only be diagnosed by finding the eggs in the feces (see p 575)

**Prognosis.**—The prognosis is bad as no cure is known.

**Treatment.**—An attempt may be made to kill the worms by thymol or Iodoquin, otherwise the treatment must be symptomatic

**Prophylaxis.**—Nothing can be said as to prophylactic measures until the method of infection is known.

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## CHAPTER LXXXIV

# DISEASES OF DUCTLESS GLANDS AND METABOLISM

General remarks—The thyroid system—The suprarenal system—The thymus system—Other ductless glands—Diabetes—Malaria perniosa—References

### GENERAL REMARKS

Of late years much work has been performed with regard to the

at the end of this chapter

### THE THYROID SYSTEM

The thyroid system consists of the thyroid gland and the parathyroids with probably the addition of the pars intermedia of the pituitary gland. The action of these organs has been the object of

these again may be applied to the thyroid gland or to the parathyroids

thymia or myotonia periodica

With regard to the factors which produce these changes in the



thyroid system McCarrison points out that they are three in number—viz—

A *Nutritional*—Defective and improper foods

B *Infective*—Insanitary surroundings bacterial and other toxins infectious disease constipation intestinal stasis and the associated toxæmias.

C *Psychical*—Fright grief worry consanguinity in marriage and heredity

He shows

two groups. In the first

places those of as endemic goitre conditions in thyroidism endemic cretinism and tetany in the descendants

In his second group he places such hyperplasias as are due to toxæmia or thyroiditis and as such he mentions simple toxæmia goitre and slight hypothyroidism as one section myxœdema as second section and Graves disease as a third section and these occurring in parents may produce much the same results in the descendants as in the first class but in this condition it will be sporadic and not endemic cretinism

Diseases of the thyroid gland are quite common in parts of the tropics with which we are acquainted Myxœdema has been seen by us in Ceylon but is rarer in the tropics than in the Temperate

is very rare in India and Ceylon

### ENDEMIC GOITRE

**Synonym**—Endemic thyromegaly

Goitre is much more prevalent in the tropics than has been realized hitherto and we have met with it frequently in Ceylon and Africa

**Climatology**—It is a cosmopolitan disease which though frequently met with in hilly districts is also in our experience quite common in low lying lands It does not appear to be associated

resent time is to confirm this view has been the previous ones of Lustig Grassi and many others tend to show that the causal agent

lives in earth and passes via potable water to man, in whose ali-



FIG 778—GOITRE IN A SINGAPORE WOMAN.

5 grains of iodide of potassium may be gradually increased. The liquor thyroidei or fresh tablets in doses of 2-5 grains combined with 10 grains of bicarbonate of soda and taken at night. The sour milk treatment has also been recommended.

**Prophylaxis**—The most important prophylactic measure is to filter and boil the drinking water when compelled to visit or live in an endemic region. The patient must also lead a life free from emotion if possible.

#### Congenital Goitre

McCarrison has pointed out that this is extremely common in certain Himalayan villages. Nearly every man and woman in these situations is goitrous and congenital goitre may be present in 60 per cent of the infants at the breast. The mothers of these children are often myxoedematous. The condition rarely calls for treatment as the victims usually die at or shortly after birth or recover spontaneously but the mother and child may be given the British Pharmacopoeia liquor thyroidei of which 1 to 2 minims may be given to the child at night.

#### Endemic Cretinism

According to McCarrison the cretinism of the Himalayas which does not show itself until about six months after birth may be divided into—

- i The myxoedematous type
- ii The nervous type

**Myxoedematous Type**—There is failure in growth, dwarfism, skeletal deformities, persistent infantile condition of the sexual organs as well

with cerebral atrophy and epilepsy.

The treatment of these conditions is the fresh liquor thyroidei (B.P.) in doses of 1-2 minims at bedtime combined with grey powder and bicarbonate of soda during the first fortnight. The dose of the liquor is gradually increased until 5-10 minims are given in a day. If the liquor is not available the dried gland in powder may be given in  $\frac{1}{2}$ - $\frac{3}{4}$  gram doses working up to 5-7 grains per diem.

#### Endemic Tetany

This is found in goitrous districts in the Himalayas where it is called *hatti fallategen* or turning of the hands. It is characterized by bilateral intermittent and usually painful spasms of the hands.

and feet and at times other parts of the body and increased ex-

is indicated above. Rickets must also be treated. The diet should consist of milk and meat should be avoided. Anemia by therapy.

## THE SUPRARENAL SYSTEM.

Malaria may cause these signs.

Chronic suprarenal suppression may be caused by malaria and is characterized by the signs of Addison's disease—viz apathy, adynamia, gastro-intestinal and nervous disturbance associated with bronzing of the skin and mucous membranes and a chronic cachexia with frequently attacks of diarrhea or convulsions and ending in coma and death.

In the tropics we have met with Addison's disease once in a European and with hemorrhage into both the suprarenal capsules twice—once in a still birth after a breech presentation in a native child and once in a case of acute suprarenal hemorrhage in a European lady. This last showed symptoms so remarkable in character that a brief description may be given.

### Acute Suprarenal Hemorrhage.

After a year's residence in Ceylon a young English lady, four months pregnant was suddenly taken ill with fever associated with an abnormally quick pulse, great tenderness above the umbilicus and pain in the small of the back on both sides. After a day's illness she died.

## THE THYMUS SYSTEM.

The normal weight of the thymus at birth is 13.26 grammes and it should increase till between eleven to fifteen months of age it

much enlarged thymus is associated with hyperplasia in the lymph glands and lymphoid tissues all over the body

Only once have we encountered a persistent thymus gland with some enlargement of the lymphatic glands and this was in a case of sudden death

### OTHER DUCTLESS GLANDS.

We are not acquainted with observations referring to the other internal secretions in the human diseases of the tropics

### DIABETES.

This disease is extremely common in the tropics but more especially in Asia and particularly in Ceylon though we have met with it in Africa

In India according to Waters it has been known since the day of the Susruta Samhita where it is called 'madhumeha' It was said to be unknown among the Chinese and Japanese but Reid has collected 207 cases in China and in Korea the complaint is called 'sweet water disease'

West says that it is rare in Hindu women but that it does occur even in Hindu widows and in general it may be said to be much more common in men than in women and is most frequently met with in the better or educated classes

The disease is the same as in the Temperate Zone, and will not be further considered here except to point out the frequency of boils and carbuncles due to it and also the occurrence of Kussmaul's coma, which may be induced by a malarial infection.

### Pentosuria

may occur  
possibility  
is on the  
osis is by  
mination

### MACIES PERNICIOSA.

Dr Ernest Black gives the following account of this disease which occurs among the aboriginal natives in the north of the State of Western Australia It has also been reported among the tribes of the Northern Territory of the Commonwealth where it is called 'living skeleton' disease

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## CHAPTER LXXXV

# DISEASES OF THE URINARY ORGANS

General remarks—Bilharziosis—Urinary amœbiasis—Oxaluria—Urinary myiasis and cantharidiasis—Chyluria—Mycological urinary tests—Test for quinine in the urine—References

### GENERAL REMARKS

RENAL disease in all its forms is frequently met with in the tropics where nephrolithiasis pyonephrosis and pyelitis with all their associated phenomena are by no means rare. Stone in the bladder is common in certain regions as is prostatic hypertrophy. But the only disease which really concerns us in this work is urinary bilharziosis caused by *Schistosoma hæmatobium*. Tumours of the bladder are not common in our experience. Prostatic abscess is met with at times. We have observed various mycoses of the genito-urinary organs due to fungi of the genera nocardia aspergillus monilia cladosporium. Native children in some parts of Africa (Sudan) suffer very often from a complaint called by the Arabs har bouh characterized by severe burning on passing urine. This condition is due to concentrated acid urine and the presence of gravel. Cystinuria is rare but we have met with a case.

In the present war a form of nephritis has been noticed in soldiers in the trenches (trench nephritis)

### BILHARZIOSIS

**Synonyms**—Urinary schistosomiasis Endemic hæmaturia Bilharzia disease

**Definition**—Bilharziosis is infection with *Schistosoma hæmatobium* Bilharz 1852 the eggs of which irritate and invade the

### BILHARZIOSIS

and Mesopotamia and in the West Indies. In Egypt according to Sandwith infection probably takes place in the early winter months when the floods have subsided.

**Ætiology.**—The cause of the disease is *Schistosoma hematobium*. The life-history is described in Chapter XXIV p 584. The method of infection is by the cercaræ penetrating the skin or mucous membranes and developing into adults which live and copulate in the portal and vesical veins while the eggs leave the body with the urine.

It occurs in any rice and at any age except infancy but is more common in males than in females due it is thought to the man washing in streams and working in the fields barefooted. According to Miss I. Good's investigations the disease is common also in young girls even in those who do not bathe and who use filtered water. It is rare in Europeans being much more common among the natives in whom it is prevalent among the working classes especially the field labourers.



11 779H PHOTOMICROGRAPHS OF *C. M. HUS* *contortus*

11 779A —MIRACIDIUM OF *Schistosoma Hematobium* X 12 IN WATER. (Photomicrograph)

**Pathology** The worms live in the p... vein but proceed to the vesicles of bladder to lay the eggs. The irritation sometimes of a very extensive nature giving rise to a sort of bilharzial granuloma—that is a tissue composed of round cells and eggs. Wadden...

the pathological changes into two classes the hypertrophic and atrophic. In the former which is more common in mucous membranes there is proliferation of the epithelium with the formation of flattened projections or papillomata while vesicles may also and according to Madlen by bursting give rise to the ulcer at times are seen.

The ova may escape from the mucosa according to Loos the aid of ulceration by working their way to within the cells into the lumen of the vesicles. Unlike with the mucous round-celled infiltration form the typical bilharzial granuloma which leads to much thickening of the wall of the vesicles and to connective-tissue formation may cause...



changes Lesions may also be found from the pelvis of the kidney to the meatus urinarius, but are most common in the bladder

**Morbid Anatomy.**—The earliest changes are found in the bladder in the formation of a general infiltration and thickening of the mucosa with bilharzial tissue. On the thickened mucosa is a layer of adherent mucus, mucus numerous eggs are found associated with vesicles which are especially marked around the trigone, and contain a whitish fluid in which there are eggs. The hyperæmic patches increase in size and thickness and the whole bladder becomes much thickened. Papillomata of all shapes and

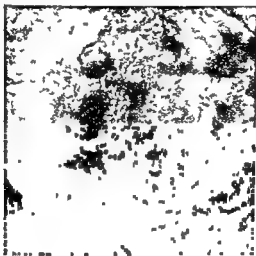


FIG 778c —BLADDER SHOWING LESIONS OF BILHARZIOSIS

sizes are formed principally on the trigone and the posterior wall of the bladder. These papillomata bleed readily, giving rise to the hæmaturia.

As the eggs increase in amount, connective tissue forms round them and they die and become calcified, thus giving rise to brownish yellow, sandy patches not protected by mucus and leading to an atrophy of the mucosa of the viscus.

The urine collects in the hollows between the papillomata and decomposing gives rise to phosphates, which produce a whitish incrustation on the bladder wall. The orifices of the ureters may

kidney. Bilharzial tissue may also form in the ureters and the pelvis of the kidney, and take the form of infiltrations and papil

omata Calculi may also form in the bladder and interstitial nephritis in the kidney

As the bladder becomes infiltrated it rises in the abdomen and

a large area of skin become involved

Bilharzial tissue may also form in the prostate and urethra

Symptomatology—The incubation is not known but according to Sandwith it varies from three to six months. At first no symp-

Loss in the legs

Sooner or later the urine begins to stagnate in the hollows in the mucosa formed by ulcers and by papillomata. When this happens the urine becomes alkaline and turbid containing pus and phosphates as well as decomposing blood and ova. With this decomposition of the urine cystitis appears and the sufferings of the patient begin in earnest with at first increased and later almost constant micturition scalding pains in the perineum and the scrotum together with tenesmus which increase until he can rest

urethra may be attacked. The urethral symptoms begin with localized pain and the formation of a lump which develops an abscess and later urinary fistula

The patient now becomes weak and anæmic and begins to suffer from pyæmia. The enlarged kidneys and at times even the ureters may be felt through the abdominal wall. In course of time septicæmia sets in and the patient dies. On the other hand, cases which have left the enfeebled area may slowly recover the ova ceasing to be passed

Complications.—Retention of urine from blocking of the urethral opening by papillomata or a stone may occur. Stone in the bladder

and cancer may also occur as a complication of the disease, but is said to be rare. Ankylostomiasis and pellagra and other diseases may also complicate a case

in small numbers

Fairley recommends a complement fixation test with an antigen prepared from livers of infected snails

**Prognosis.**—The prognosis depends largely upon the possibilities of infection, and is therefore better if removal from the infected area is possible, for, according to Sandwith, most cases cease to pass eggs within three years of leaving that area



FIG 778D — EGG of  
*Schistosoma haematobium* IN URINE  
(Photomicrograph)

**Treatment.**—McDonagh and Christopherson have recommended the intravenous injection of tartar emetic which should be administered in the same way as for intestinal schistosomiasis (p 1869). Various treatments such as injections of sulphuretted hydrogen and carbon dioxide gases into the bladder, have been suggested. The drug commonly used in the past was liquid extract of male fern in 5 minims doses, three times a day continued for a long time. It is said to reduce the hæmaturia and lessen the discharge of eggs. Emetine has been recommended by several observers. Madden recommends the washing out of the bladder with injections of silver nitrate, beginning with 1 in 10,000 and increasing the strength gradually, or quinine in a 4 per cent solution, or adrenalin in normal saline solution

three times a day or day, urotropine, calol, buchu and hyoscyamus mixture may be administered. Large quantities of water or Vichy water should be drunk to wash out the urinary passages

diarrhoea and septic infection set in, and the patient dies in about two weeks

With regard to fistulae, they ought to be thoroughly dissected out and this may mean a very prolonged operation if it is to do any good

McDonagh who first used antimony in balharziosis in 1912 prefers colloidal antimony which he gives intravenously and intramuscularly in doses of

nly  
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with some disinfectant before being disposed of

### URINARY AMOEBIASIS

**Definition**—Urinary amoebiasis is the infection of the urinary tract with amoebae which most commonly are *Loeschia histolytica* (Schaudinn 1903)

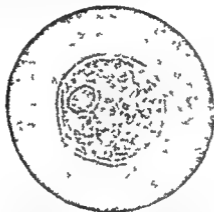


FIG. 779.—PRECYSTIC STAGE OF *Loeschia histolytica* (SCHAUDDIN 1903) AS SEEN IN URINE IN A SUDAN CASE. FRESH AND UNSTAINED SPECIMEN (X1500 DIAMETERS)

(Microphotograph. This illustration may be examined with advantage by means of a reading lens.)

**Historical**—In considering the history of urinary amoebiasis it is convenient to divide the subject into infections low down in the urinary tract into those which are situated high up and into those

and blunt pseudopodia possessed granular cytoplasm containing

a vesicular nucleus and phagocytéd red blood-corpuscles, while cystic stages of the same organism were also observed. There is, in our opinion, no reason to doubt that this is the same species as the

*dysenteriae* Councilman and Lafleur, 1891; and *Amœba coli* Loesch, 1875, and by various other names

The unpleasant point is that if the rules of zoological nomenclature are pressed we ought to call the amœba of dysentery by Loesch's name.

(2) In Jurgens' 1892 case the amœbæ were found by post-mortem examination to come from mucous cysts in the bladder of an old woman who had suffered from cystitis, and in whose vagina they were also found

(3) In 1911 Craig found *Loeschia histolytica* Schaudinn, 1903, in an infection of the bladder in which the autopsy showed a minute fistula between the ulcerated intestine and the bladder

(4) Lynn's 1914 Costa Rica case seems to have been probably caused by a vesical lesion, as the patient is reported to have felt 'a pain in the trigonum vesicæ at the end of micturition'. He responded very well to emetine treatment, as it is stated 'in the course of five days the vesicle tenesmus was relieved and the urine cleared'; moreover, there was no return of the symptoms after one month

(5) and (6) Scott Macfie's second case was probably a bladder infection associated with bilharziosis, while his third and fully

symptoms has been recorded by Chalmers and O'Farrell in the Anglo-Egyptian Sudan.

*Site in the Urinary Tract Unknown.*—(9) We have been unable to refer to the original papers written by Wijnhoff, by Jeffries, and

## OXALURIA

by Fisher, and are therefore unable to state where the infection was situated.

(10) The very brief note by Ward Coles and Friel arouses the doubt as to whether the amoebæ really came from the patient because they do not state whether these bodies were merely seen once or whether they were of frequent occurrence. They call the organism *Amoeba urinae granulata* but as Fartham has pointed out, in no case can this name stand.

(11) Scott Macfie, owing to the non return of the patient to the hospital was unable to define the site of the infection in his first case. All the cases which we have met with have been associated with symptoms which have pointed to the pelvis of the kidney as the probable source of infection.

**Ætiology**—It is possible that all reported cases were due to *Loeschia histolytica*.  
**Symptomatology**—*Amoebic Pyelitis*. With or without the history of previous amoebiasis in the form of amoebic dysentery or other intestinal infection a person is seized with an attack of lumbar pain and slight fever, the temperature rising to 99-100°. The urine if collected and examined after centrifuging will be found to contain a deposit of red blood cells, leucocytes and amoebæ in the precipitate stage and more or less degenerate. The blood when examined has been found to show

Polymorphonuclear leucocytes	614
Mononuclear leucocytes	84
Large lymphocytes	224
Small lymphocytes	56
Mononuclear leucocytes	22
Mast cells	14
Total	1000

**Amoebic cystitis**—In these cases there is pain and strain at the end of micturition. On examination the urine shows red amoebæ.

**Treatment**.—The treatment is to give urotropicine by the mouth or time intramuscularly.

## OXALURIA

**Definition**.—Oxaluria is the deposit in abnormal quantities of oxalate crystals in the urine and should be restricted to those cases in which show an increase in the quantity excreted in the day.

**Historical**.—The crystals of calcium oxalate were discovered by Dumas in 1838 and were much discussed for a time being regarded as the basis of the oxalic acid diathesis of Prout. Gellinger and Bird attributed it to a result of Smoler and Bacon's investigations but fell to the ground. In 1896 Darlow attributed them to a metabolic defect. In 1900 Baldwin conducted a series of experiments in 1900 Baldwin conducted a series of experiments

■ vesicular nucleus and phagocytéd red blood-corpuscles, while cystic stages of the same organism were also observed. There is in our opinion, no reason to doubt that this is the same species as the organism.

(Schaudin

Schaudin

*dysenteriae*

1875, and by various other names

The unpleasant point is that if the rules of zoological nomenclature are pressed we ought to call the amœba of dysentery by Loesch's name

The following cases are known to us —

(1) Baelz's patient was dying from pulmonary tuberculosis, and it

(2) In Jurgens' 1892 case the amœbæ were found by post-mortem examination to come from mucous cysts in the bladder of an old woman who had suffered from cystitis, and in whose vagina they were also found

(3) In 1911 Craig found *Loeschia histolytica* Schaudinn, 1903, in an infection of the bladder in which the autopsy showed a minute fistula between the ulcerated intestine and the bladder

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He responded

the course of

infection associated with diarrhoea, while his case was probably described as probably an infection of the genito-urinary tract

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granular bodies 50 by 28 microns, which slowly altered their shape and which contained, among other things, red blood-corpuscles and

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*Site in the Urinary Tract Unknown*—(9) We have been unable to refer to the original papers written by Wijnhoff, by Jeffries, and

## OXALURIA

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(11) Scott Macfie owing to the non return of the patient to the hospital was unable to define the site of the infection in his first case. All the cases which we have met with have been associated with symptoms which have pointed to the pelvis of the kidney as the probable source of infection

**Climatology**—Cases are known in Europe Africa and Asia

**Ætiology**—It is possible that all reported cases were due to *Loeschia histolytica*

**Symptomatology**—*Amœbic Pyelitis*—With or without the history of previous amœbiasis in the form of amœbic dysentery or other amœbic infection a person is seized with an attack of lumbar pain and slight fever the temperature rising to 99° 100° F

The urine if collected and examined after centrifuging will be seen to contain a deposit of red blood cells leucocytes and amœbe in a precystic stage and more or less degenerate

The blood when examined has been found to show —

Polymorphonuclear leucocytes	61.0
Mononuclear leucocytes	8.4
Large lymphocytes	22.4
Small lymphocytes	5.0
Leucophagocytic cells	2.2
Mast cells	0.4
Total	100.0

**Amœbic Cystitis**—In these cases there is pain and strain at the end of micturition. On examination the urine shows many amœbe

**Treatment**—The treatment is to give urotropine by the mouth and emetine intramuscularly

## OXALURIA

**Definition**—Oxaluria is the deposit in abnormal quantity of oxalate crystals in the urine and should be restricted to those which show an increase in the quantity excreted in the day

**Histories**—The crystals of calcium oxalate were discovered by Donne in 1838 and were much discussed for a time being based on the oxalic acid diathesis of Prout Golding Bird and others but as a result of Smoler and Bacon's investigations they fell to the ground. In 1896 Dunlop attributed them to the food and in 1900 Baldwin conducted a series of experiments



in vesicular nucleus and phagocytosed red blood-corpuscles while cystic stages of the same organism were also observed. There is in our opinion no reason to doubt that this is the same species as the organism

(Schaudin " "

Schaudin

*dysenteriae* "

1875 and by various other names

The unpleasant point is that if the rules of zoological nomenclature are pressed we ought to call the amœba of dysentery by Loesch's name

The following cases are known to us —

(1) Baelz's patient was dying from pulmonary tuberculosis and it

(2) In Jurgens' 1892 case the amœbæ were found by post mortem examination to come from mucous cysts in the bladder of an old woman who had suffered from cystitis and in whose vagina they were also found

(3) In 1911 Craig found *Loeschia histolytica* Schaudinn 1903 in an infection of the bladder in which the autopsy showed a minute fistula between the ulcerated intestine and the bladder

(4) Lynn's 1914 Costa Rica case seems to have been probably caused by a vesical lesion as the patient is reported to have felt a pain in the trigonum vesicæ at the end of micturition. He responded very well to emetine treatment as it is stated in the course of five days the vesicle tenesmus was relieved and the urine cleared. Moreover there was no return of the symptoms after one month

(5) and (6) Scott Macfie's second case was probably a bladder infection associated with bilharziosis while his third and fully described case was probably an infection of the genito-urinary tract

infection

passage

1 red and

and large

granular bodies 50 by 28 microns which slowly altered their shape

— loc and

symptoms has been recorded by Chalmers and O'Farrell in Anglo-Egyptian Sudan

*Site in the Urinary Tract Unknown*—(9) We have been unable to refer to the original papers written by Wijnhoff by Jeffries and

y Fisher, and are therefore unable to state where the infection  
is situated.

(10) T  
doubt as  
because  
ence or

organism *Amœba urinae granulata* but as Pantham has pointed out  
in no case can this name stand

probable source of infection

Climateology —Cases are known in Europe Africa and Asia

Ætiology —It is possible that all reported cases were due to  
*oeschia histolytica*

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emetine intramuscularly

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Definition —Oxaluria is the deposit in abnormal quantity of  
oxalate crystals in the urine and should be restricted to cases  
which show an increase in the quantity excreted in the day

Etymology —

upon dogs and showed that fermentative intestinal disturbances associated with the absence of free hydrochloric acid in the gastric

With regard to its presence in natives Burkitt's observations in Chandra in Eastern Bengal are especially interesting

**Ætiology**—The normal quantity of calcium oxalate to be excreted in the urine is 0.5 gramme per diem. It may perhaps come

Chandra in Eastern Bengal and consuming nothing but vegetables and fruit suffer excessively from oxaluria. Certainly treatment based upon the idea of too little acidity in the stomach is most successful.

**Symptomatology**.—Dyspepsia, pain in the lumbar region shooting down the ureter, burning during micturition and even hæmaturia are signs of oxaluria. These symptoms may or may not be

or bladder.

**Treatment**—The treatment is simple and certain. A few minims of the dilute pharmacopœial preparation of nitrohydrochloric acid combined with  $\frac{1}{2}$  drachm of the compound tincture of cinchona bark and 1 ounce of the compound mixture of gentian given three times a day before meals is the best remedy.

### URINARY MYIASIS AND CANTHARIASIS

See Chapter LXVII pp 1628 and 1640

### CHYLURIA.

This is generally due to filariasis and is described on p 1608. Cases of schistosomal origin occur and Remlinger has placed on record two cases due to hydatids with hydatid membrane in the urine.

### URINARY TESTS

Castellani and Taylor's *Mycological Method for the Detection of Glucose, Lactose, Maltose, and Other Carbohydrates in the Urine*.—The tropical practitioner generally bases the diagnosis of diabetes on the reduction of Fehling's solution by the suspected urine. Fehling however may be reduced by a

number of other substances in addition to glucose—for instance by lactose

## FORMULAS —

## URINE FEHLING REDUCING

1	<i>Monilia baltanica</i> Castellani	Gas	=glucose
2	<i>Monilia baltanica</i> Castellani	■	} =levulose
	<i>Monilia krusei</i> Castellani	Gas	
3	<i>Monilia krusei</i> Castellani	O	} =maltose
	<i>Monilia pinoyi</i> Castellani	Gas	
4	<i>Monilia pinoyi</i> Castellani	O	} =galactose
	<i>Monilia metalonidensis</i> Castellani	Gas	
5	<i>Monilia metalonidensis</i> Castellani	O	} =pentoses
	<i>Bacillus coli sensu stricto</i> Escherich	Gas	
	<i>Bacillus paratyphosus B</i> Shottmüller	Gas	
6	<i>Bacillus coli</i> Escherich	Gas	} =lactose
	<i>Bacillus paratyphosus B</i> Shottmüller	O	

## URINE NOT FEHLING REDUCING

O	} =saccharose
Gas	
O	} =saccharose
Gas	
Gas	} =inosite
O	

## URINARY TEST FOR QUININE ELIMINATION

At times it is necessary for the physician to be certain that quinine is being absorbed by the patient and at others that the patient is really taking the quinine which has been ordered. Under such conditions a simple easy

eight hours after that of 20 grains of the drug

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## Urinary Tests.

## CHAPTER LXXXVI

# DISEASES OF THE GENERATIVE SYSTEM

General remarks—Male generative system—Endeemic funiculitis—Female generative system—Tropical puerperal fever—Sutika—References—Addendum—Ante- and post natal pathology—Addendum references

### GENERAL REMARKS

In this chapter we propose to include a few general remarks upon the male and female generative systems. The subjects will be merely touched upon as space forbids anything else.

### MALE GENERATIVE SYSTEM

excess is distinctly more deleterious in the tropics than in temperate  
boys appears at an

Eugenics and have mentioned the ill effects of *alcoholism syphilis*  
upon the subject of  
*gonorrhœa* and *tuberculosis* the effects of which are much the same  
as in other climates but as regards the first we have set forth

observed in temperate zones but complications are much more

believe it is contracted by passing water on the ground illuminated  
by moonshine or by passing water where dogs have previously  
urinated. Occasionally true cases of non gonorrhœic urethritis are

*chyloceles*, *hematoceles*, *orchitis* and *epididymitis* are all common and bilharziosis has been already noted by us. Epithelioma of the penis is associated with phimosis and the accumulation of irritating secretions which may cause balanitis. In these circumstances, if no treatment is carried out epithelioma may result. Certainly epithelioma of the penis is rare in races in which circumcision is performed.

and testes—and as may be imagined but few survive such a mutilation performed on the field of battle. The result of these opera-

### ENDEMIC FUNICULITIS

**Synonyms**—Suppurative phlebitis of the spermatic cord. Sup-

*puritis cordis*.

**History**—For many years medical men practising in Ceylon have noticed the occurrence of a peculiar form of acute suppurative inflammation of the spermatic cord which occasionally takes a true epidemic character, numerous cases occurring within a short period. References to this affection may be found in almost all the medical reports for the colony during the last twenty years, the disease being variously indicated by the name of phlebitis of the cord or corditis. It was considered by some to be of traumatic origin, by others of venereal origin. Some practitioners consider it



it to be a malarial affection. The disease was investigated by Castellani first in 1904 and more completely in 1907. He came to the conclusion that it had nothing to do with either malaria or gonorrhœa. He thought the malady had sufficiently characteristic symptoms to be ranked as a separate disease and suggested the name endemic funiculitis. The affection is not limited to Ceylon

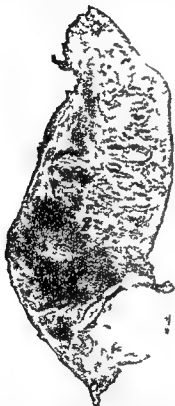


FIG 780—ENDEMIC FUNICULITIS SHOWING LONGITUDINAL SECTION OF THE CORD

The testes and epididymis are normal



FIG 781—TRANSVERSE SECTIONS OF THE CORD IN ENDEMIC FUNICULITIS

The testes and epididymis were normal but the tunica vaginalis held a small quantity of fluid. The sections of the cord are arranged in an arch from right to left. The dilated veins which were filled with pus are clearly visible.

as cases occur in Southern India and an identical pathological condition has been described in Egypt by Madden in 1907 under

**Ætiology**—Among Ceylon practitioners the disease was considered by some to be of traumatic origin others believed it to be of venereal origin and yet others to be a malarial affection. Castellan found in all the cases virulent diplo-streptococci and in some

while they are Gram positive in cultures. According to Coutts these micrococci are often found in the urethra of natives. He regards the suppurative condition of the cord as due to the extension of an infection from the urethra by way of the vas deferens. Wise has found in his cases *F. bancrofti* and numerous streptococci. Piester believes the disease to be connected with bilharziosis. We are inclined to consider the malady to be a filarial condition with a superadded streptococcus infection. The filaria probably plays the more important or only rôle in the subacute or



FIG. 782.—TRANSVERSE SECTION INFLAMED CORD (ACTUAL SIZE)

tance must be given to a sudden chill or to some form of traumatism. In nearly all the Ceylon cases the disease begins abruptly after taking a cold bath when feeling tired. In some cases the patient gives a history of having made

extends to the epididymis. In two very recent cases of ours however the epididymis was normal. The testicle proper remains

**Morbid Anatomy**—At the post mortem examination of the cases in which an operation has not been performed in time the lesions found are those of a septicæmic process. The skin is jaundiced and may present petechiæ. The lungs often show hypostatic congestion.

often congested

**Symptomatology**—The disease begins suddenly generally after a hard day's work or severe exercise. In Ceylon the usual history is as follows. The patient after an extra hard day's work comes home in the evening very tired but not feeling unwell and takes a



FIG 783.—ENDERMIC FUNICULITIS  
IN A SINHALESE MAN

cold bath as usual. After the bath he is suddenly seized with a shivering fit, the temperature rising very high. He feels very sick and there is often actual vomiting. At the same time he complains of pain along the cord and the epididymis. The condition becomes rapidly worse and the patient is generally taken to hospital on the second or third day of the illness. On admission it is usually found that the general condition is grave. There may be continuous vomiting and occasionally hiccough. The temperature is generally above 102° F and the pulse small and frequent. At the physical examination it will be seen that the inguinal region is occupied by a large cylindrical swelling in the direction of the cord.

The swelling is very tender on pressure and hard. The skin is not affected. Generally the epididymis is somewhat enlarged and tender though in some very recent cases it may not appear to be affected. In all cases the testicle proper appears to be normal.

The affection attacks both sides. The ulcers will be made to spontaneous recovery. If an operation is not performed in time signs of general septicæmia usually set in. In such cases the skin of the patient often becomes jaundiced. Cutaneous hæmorrhages

may appear, the fever is of an intermittent or remittent type, the pulse becomes extremely small, there is often hiccough, and the

operation is not performed in time, symptoms of general septicæmia

in some cases, especially in the tumbling form, operative treat-

ichthyol ointment with or without ice application may be sufficient,

## NON-GONORRHOICAL URETHRITIS

Urethral mucopurulent discharge is as a rule of gonorrhœal origin, but Castellani has called attention to a number of forms of quite different ætiology, which may be classified as follows —

### A. Traumatic Mucous Urethritis

This condition is generally caused by the use of a hard catheter or by the

### B. Urethritis of Hyphomycetous Origin.

The following clinical varieties may be differentiated —

1. The discharge is black, greenish black, or brownish black, generally due

fus  
sis  
or  
nt

C Urethritis associated with Animal Parasites. I With Protozoa, II With  
Animal Parasites Higher than Protozoa

I Urethritis of Protozoal Origin.—The following varieties may be distinguished —

(4) *Spirochaeta Urethritis*—This type of urethritis due to *S. urethralis*

genus *Monilia*

FEMALE GENERATIVE SYSTEM

years undergoes one of two varieties of this operation. The milder operation called *sunna tahuret* or circumcision according to religious law consists of the removal of the clitoris and labia minora while the more severe cutting called *Pharaoh's tahuret* or the old

nces—e.g.  
lofan and  
ve or six

birth

In 1910 Wilson studied the peculiar elongation of the nymphæ found in Hottentot women and came to the conclusion that it was largely produced artificially

In 1917 Neve drew attention to the fact that rickets is rare in Kashmir but that osteomalacia was common in multiparous

## VULVO VAGINITIS

## VAGINAL PROTOZOA

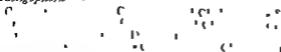
The vagina of native women may be the habitat of numerous protozoa apart from *Treponema pallidum* and spirochaetes. We record the presence of the following —

*Sarcodina* —

*Loeschia histolytica* (Schaudinn 1903)

*Loeschia coli* Loesch 1875

*Mastigophora* —



*Ciliata* —

*Balantidium vaginale* Castellani and Chalmers 1918

### TROPICAL PUERPERAL FEVER

**Synonym.**—Puerperal septicæmia

**Definition.**—Tropical puerperal fever is an infection of parturient or puerperal women with various germs which may cause a local

has been prevalent an has roamed but as it is conveyed from infective sources living or dead autogenetic or heterogenetic by instruments or by the hands of the attendants to the uterus of the parturient woman it is obvious that, in those

and con

n but may the child the fact iving help even if her

often to be rendered to the parturient woman from time immemorable and that puerperal fever in isolated cases and in epidemics has been known for ages

topic—viz that the researches of e various peoples inhabiting that island

These inquiries showed that the deaths of women in childbirth were higher than those in Europe and that the principal cause was puerperal fever





we have found streptococci and other organisms. These people are well acquainted with puerperal fever, which they call 'el jarat' or sometimes 'humma nafas' or when slight they term it milk fever or 'humma laban' and which they consider to be transmitted from one case to another.

The civilized peoples of Khartoum and Omdurman more especially the Greeks, Syrians and better class natives suffer from *febris puerperalis* and *febris in puerperio*, but no statistics are available to show the incidence of the disease.

As regards Zanzibar the deaths from puerperal fever are given as eight out of a total mortality of 1022 of which 572 are female deaths at all ages.

In none of the above statistics is it possible to compare the puerperal deaths with the number of births, as these were not accurately known.

The mortality and morbidity from puerperal sepsis and the infantile death rate among native communities are enormous.

He blames the village midwife or handywoman who he says is a prejudicial ignorant, and dirty person and summarizes his remarks by stating —

He blames the village midwife or handywoman who he says is a prejudicial ignorant, and dirty person and summarizes his remarks by stating —

'We see that for the native woman under her native skies want and poverty may play havoc with the child that is to be but there is often a physical environment of ignorance and sepsis during labour which demands the attention of public opinion.

Our experience in various tropical and subtropical regions

there appeared the autogenetic and the microbic in origin

In 1788 Denman observed that the disease was carried from cases of puerperal fever to healthy lying in women by doctors and mid-

work of Alexander Wendell Holmes in asserting that not that it could come

in a similar way from a case of erysipelas or from a post mortem and that it was necessary for the physician to disinfect his hands

case of puerperal fever as a sound basis by Sem we unknown to require

recapitulation

of the disease is d

and Mackay have revived *S. puerperalis* but Arloing's name has

1898 found it in the secretions of the normal vagina as well as in pus from Bartholin's glands and in the exudate of retained placenta. It was next observed by Jeannin in 1907 to be present in numerous cases of putrid puerperal infection, and is thought by Veillon's pupils to be the same organism as the anaerobic streptococcus found in vaginal secretion in 1897 by Menge and Kroenig, whose work had been questioned by Koblank but supported by Natvig, Schottmuller, and Hamm.

In 1907 Gioelli reported the presence of a coccus, thought to be a staphylococcus which he found in a peri-uterine abscess and named *Coccus anaerobius* Gioelli 1907 while in 1908 he dealt with the

puerperal fever but also in otitis media, meningitis, cysto-pyelitis, abscess of the lung, gangrene of the lung and empyema, and he

severe and fatal cases —

*S. erysipelatos* Fehleisen 1883 in fifteen cases in the uterine discharges and  
 Schottmuller, 1910,  
*S. erysipelatos*  
 both together in

He also met with *Streptococcus mitior* seu *viridans*, an aerobic organism first described by himself in 1903, in the uterus and blood of one case.

He considers that there are two distinct methods of infection, the

contagious

The second method of infection is the heterogeneous caused by *S. erysipelatos* and brought from an external source of infection to the puerperal woman as just described

Van L. ... .. deduced that the importance of

appears to us that there is a general agreement between Schott muller's organism and that described by Veillon. They both

the body. They are probably the same organism as the anaerobic vaginal streptococcus described by Menge and Kroenig and also that found by Gioelli in 1907

All these organisms in our opinion should be classified under the name of *S. fetidus* Veillon 1893

In 1901 Lewkowicz found an obligatory anaerobic streptococcus which he named *S. anaerobius micros* in the mouths of sucklings. In 1907 Jeannin reported that it was present fairly frequently in puerperal infections. It is described as being lanceolate and usually disposed in diplococcal forms and only occurring in short chains.

In 1812 Furneaux Jordan published an important lecture upon 'Puerperal Infection'. He and Mackay examined the uterine discharges of twenty one cases of puerperal fever and found streptococci in seventeen cases—i.e. 80 per cent. This streptococcus was identical in all cases and was said to be quite distinct from other streptococci and so the name *Streptococcus puerperalis* Furneaux Jordan and Mackay 1912 was given to it but we have noted that Arloing had already applied this name in 1884 to a streptococcus which he obtained from cases of puerperal fever and which he believed to be distinct from *S. erysipelatos* and *S. pyogenes* the only named forms at that time but at present all three are considered

that *S. salinaris*, *S. bovis* were causal germs in the origin of these germs to

human saliva (*S. salinaris*) zibla—i.e. horse-dung used for walls and floors—bovine faeces and equine faeces etc (*S. bovis* and *S. versatilis*)

therefore —

T

1

2

3

- 4 *S mitior* Schottmuller 1903
- 5 *S puerperalis* Furnerux Jordan and Mackay 1912
- 6 *S salivarius* Andrewes and Horder 1906
- 7 *S bovinus* Broadhurst, 1915 (synonym *S bovis* Chalmers and Atiyah 1916)
- 8 *S versatilis* Broadhurst 1915

is still unnamed.

*Abortion*—A very interesting point is the question whether the same organ

**Methods of Infection**—Adverting to the methods of infection of the parturient woman we note that Geddes in his *Statistics of Puerperal Fevers* published in 1912 says that he believes 99 per cent of the cases are due to those conducting the labour. A statement of this nature makes it imperative to possess some knowledge as to the presence or absence of bacteria in the normal vulva, vagina and uterus.

There has been great interest in the question of the

The secretion of the normal uterus is by a consensus of opinion considered to be sterile

than the latter

With regard to the latter the researches of Chalmers and Atiyah have thrown suspicion upon cow and horse dung as sources of infection and this has been confirmed by the work of Chalmers and Marshall who found the same organisms in bovine and equine faeces

There are three native uses of cow and horse dung and they are —

As this wash dries it must form dust which must pollute the air and be driven hither and thither by the strong winds which are

of the attendants on the parturient woman and so bring about the infections may be kept up indefinitely by the same means

**Pathology** —The post mortem anatomy shows some of the features well known in Europe

**Symptomatology** —There seems to be no doubt that the same organisms can cause the mild febris in puerperio and the severe febris puerperalis.

Why there should be such a difference is not clear and must depend in some way or another upon the general bodily condition of the patient and perhaps upon the strength of the streptococcal strain which as is well known from laboratory experiments varies considerably

in puerperal fever in most as already stated remaining low or sinking in serious or fatal cases but rising as improvement sets in

**Diagnosis** —It is of the utmost importance that any fever attacking a puerperal woman in the tropics should be assumed to be puerperal fever until it is proved to be something else

The presence of one of the varieties of puerperal fever can be confirmed —

With regard to the differential diagnosis the most important fever which requires attention is malaria occurring in the puerperium and this should be capable of easy differentiation by —

1 An examination of peripheral blood smears for the parasites or if they cannot be found by—

2 A differential leucocytic count with the discovery of a distinct mononucleosis which cannot be explained by other protozoal infections such as amœbic dysentery kalī azar etc

3 Enlargement of the spleen not due to one of the forms of tropical splenomegaly

If these three tests fail to decide the presence or absence of malaria then a few doses of quinine should be administered and can do no possible harm and may even benefit the patient if a puerperal infection due to streptococci or bacteria is present. In our

## ADDENDUM

## REMARKS ON ANTENATAL AND POSTNATAL PATHOLOGY

General remarks—Antenatal pathology—Postnatal pathology—References

## General Remarks

We think that perhaps a few remarks concerning antenatal and postnatal pathology may be of interest

## Antenatal Pathology

Much useful work has been done of late in Calcutta and other tropical towns by means of *lady health visitors* by the training and provision of *midwives* as well as by the training of school teachers and school girls in matters connected with hygiene. The

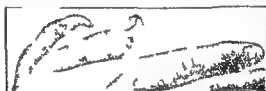


FIG 784.—POLYDACTYLISM

effect of skilled attention at the time of birth has been indicated by the entire absence of *tetanus neonatorum* amongst babies delivered



FIG 785.—CYCLOPS  
(From a photograph by f)

by the municipal m d w  
low death rate under I  
life as related by Mts. D,

a v

extraord narily  
first week of

to combat such

given if necessary as so many monstruosities do appear in the tropics that some care is required to diminish their numbers



FIG. 786.—ISCHIOPAGUS TRIPUS

Since the days of Læcetus many attempts have been made to classify monstruosities by such authorities as Buffon Blumenbach

1 of Bischoff Goerster Fischer Ahlfield and others while

t and

**Hemiterata.**—Anomalies of volume form colour situation & position number and existence

**Heterotaxis.**—Splanchnic inversion and general inversion

**Hermaphrodites.**—True including bilateral unilateral and lateral and false —: with double sexual external genitalia but unsexual glands



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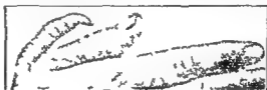


FIG 784.—POLYDACTYLISM

effect of skilled attention at the time of birth has been indicated by the entire absence of *tetanus neonatorum* amongst babies delivered



FIG 785.—CYCLOPS  
(From a photograph by Sambon)

by the municipal midwives in Calcutta and by the extraordinarily low death rate under similar conditions during the first week of life as related by Mrs. Lewis.

numbers

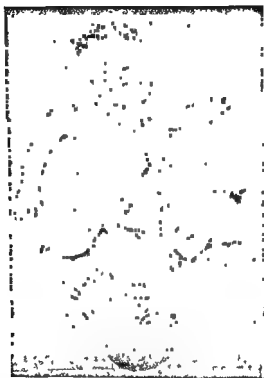


FIG 786—ISCHIOPAGUS TRIPUS

Since the days of Licetus many attempts have been made to classify monstrosities by such authorities as Buffon Blumenbach Meckel Bischoff Foerster Fischer Ahlfield and others while Ballantyne has written a most interesting book on the subject

We have always used the classification introduced by Hirst and Pierson in 1892 and have found it useful. It is as follows—

**Hemiterata.**—Anomalies of volume, form, colour, structure disposition, number, and existence

**Heterotaxis.**—Splanchnic inversion and general inversion

**Hermaphroditis.**—True including bilateral unilateral, and lateral and false—*i.e.* with double sexual external genitalia, but unisexual glands

The great danger of this period is diarrhœa or dysentery from infections with amœbæ or bacilli

All the diseases which attack adults in the tropics may affect the child after birth and the special features which they show have already been recorded as well as the treatment in the chapters on the various diseases and need not be repeated but the danger of the child being infected with tuberculosis is very great especially in the slums of large towns

**Dosage of Drugs**—As we have often been asked for the dosage of drugs in a child we give the following rules—Several

age is Cowling's rule with Brunton's modification

In brief it is to take the full adult dose and divide it by a factor obtained

four divided by the child's age in years  
 over twenty

suppose that the full adult dose is 6 grams and the child's age next birthday is four years then the factor is  $\frac{4}{24}$  (i.e.  $\frac{1}{6}$ ) and the dose is 1 grain.

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## CHAPTER LXXVII

# DISEASES OF THE LYMPHATIC SYSTEM

General remarks—Climatic bubo—Volvulus—References

### GENERAL REMARKS

area—e.g. a gonorrhœal salpingitis—are all met with Chylous  
abscesses is rare

### CLIMATIC BUBO

**Synonym**—Glandula idiopathica (Brooke)

**Definition**—Climatic bubo is the enlargement of the inguinal

**Climatology**—The chief geographical distribution of climatic bubo is the East Coast of Africa the West Indies the Straits Settlements and China but it may be met with in any tropical

and subtropical region. Le Dantec quotes cases occurring in Mauritius, Tonkin, and Madagascar. Low and Castellani have described a case in Uganda, while Luzzatti has met with several cases in Chili. We have observed cases in Ceylon, and Skinner has described a case in Bengal. Climatic bubo may also come across several other Mediterranean

districts.

**Ætiology.**—Though various micro-organisms have been described, the ætiology of the disease is still obscure.

In our opinion, climatic bubo is a disease *per se*, and is not related to plague.

**Predisposing Causes.**—The condition is apparently most commonly met with in sailors and stokers. It occurs chiefly in young adults, and is said never to be found in children.

**Pathology.**—The results of our histological researches agree better with those obtained by Vanzetti in Italy on the material collected in South America by Luzzatti than with those of other authors.

The capsule of the affected gland is much thickened, and the

shown by the classical histological investigation of Duerck, plasma cells are absent or extremely rare, while the so called retractile

is not known  
Ley and Rost  
set is generally

## PATHOLOGY -- DIAGNOSIS

gradual, after perhaps two or three days of vague malaise and slight fever the patient complains of pain in one or both of the inguinal regions which increases on walking. On examining the parts both sides or more frequently one side only will be found to be enlarged very painful on pressure, and hard. The swelling may be as large as a goose's egg. There will be no signs of lymphangitis present. The glands may become greatly enlarged reaching the size of a hen's egg or larger but in most cases do not suppurate. Aspiration by means of a syringe will draw only a little gland juice occasionally blood stained but no pus. The liquid will be found to be sterile.

Fever is often present of an irregularly remittent or intermittent type. It rarely exceeds 102° F and is higher at night than in the morning. The duration of the disease is variable from a few days to several weeks and rarely months. In time the pain subsides and the size of the enlarged glands returns to normal.

Occasionally a relapse occurs though the patient may be very weak and unfit for work. A slight leucocytosis. The examination of the blood may reveal normal. Occasionally a trace of albumen may be present. Intestinal symptoms are generally absent.

**Clinical Varieties**—In our experience an acute and subacute and chronic type of climatic bubo may be distinguished. The acute type is always accompanied by fever and severe pains in the affected glands. All the symptoms disappear within five to ten days. In the subacute and chronic type lasting from a few weeks to several months the fever may be absent altogether. Moreover in some cases the periglandular tissues become inflamed suppurate and may take place and fistulous tracts may develop.

**Diagnosis**—The absence of soft chancre on the genital organs will exclude venereal bubo. The absence of lymphangitis infection wounds insect bites on the legs and feet will exclude the ordinary bubo. In contrast to plague the patient does not have a characteristic bubo. In contrast to plague the patient does not have a characteristic bubo. In contrast to plague the patient does not have a characteristic bubo.

To distinguish between the different types of the affected gland with certainty or certainty part of the gland juice which is drawn off is then examined microscopically and by culture method in the ordinary way for the presence of the plague bacillus.

fil  
fe  
of

sterile will clear the diagnosis

**Prognosis**—The prognosis is favourable though in some cases the affection may last for several months. A relapse may occasionally occur shortly after the first attack is over sometimes on the side previously affected sometimes on the other side.

**Treatment**—The treatment is merely symptomatic consisting of complete rest application of lead lotion on the affected region or an ichthyol and belladonna ointment together with the administration of a mild aperient and if there is much pain a hot fomenta

has been recommended by G. Rost

### VOLVULOSIS

**Definition**—Volvulosis is a disease caused by *Onchocerca volvulus* Leuckart 1893 and characterized by the formation of fibrous cutaneous or subcutaneous tumours.

**History**—As already mentioned on p. 649 these tumours were discovered by a German medical missionary to contain worms which were described by Leuckart in 1893 and subsequently the disease and its causative worm were studied by Labadie-Lagrave and Deguy in 1899 Prout in 1901 Bump in 1904 Ziemann and Védy in 1907 and Fulleborn in 1908 the last paper being a most valuable contribution to our knowledge of the subject.

**Chmatology**—The disease is found in Africa at Sierra Leone on the Gold Coast and in Dahomey Cameroons and the north east of the Congo where it is variously stated to affect from 1 per

1893 but the method by which it enters the human body is quite unknown as is also its life cycle outside

human beings. Brumpt has however suggested that it will probably be found in a tsetse-fly because of its riverine distribution and the fact that microfilariae have been found in the peripheral circulation by Fulleborn, Rodenwilt and others.

**Pathology**—The adult worms lie in lymphatics the anterior end of the female being in close approximation to the posterior end of the male because the apertures of the genital apparatus lie in these positions. By some means—possibly by the presence of the possibly by toxic waste-products—these worms

cause inflammation of a portion of the lymphatic system and which is associated with the formation of a tumour and female worms and *Microfilaria* etc.

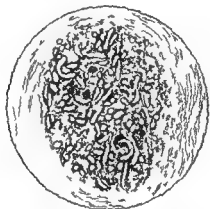


FIG. 788.—TUMOUR CONTAINING *Onchocerca volvulus* LEITCH (After Fulleborn.)

On opening one of these tumours it is seen to consist of a capsule of connective tissue beneath which is some soft caseous material containing granular debris and Microfilariae. The tumour is ever present in the cavity of the lymphatic vessel.

Microfilariae  
irregular fever but it is said that



may be noticed. It is

discovered in the sub-  
the chest, the region  
ours vary in size from  
subcutaneous tissue,  
may be elastic if they  
fibrous tissue. They

usually remain quiescent for years, and but seldom ulcerate.

Bernard and Ouzileau believe that *Onchocerca volvulus* may also give rise to true elephantiasis in certain parts of Africa.

**Diagnosis.**—The presence of an elastic tumour somewhat resembling a lipoma, or of a firm fibrous tumour in any part of the body of a person who has resided in the endemic region should arouse suspicions as to the presence of *Onchocerca volvulus*.

When the nodules are in the proximity of articulations the condition may closely resemble the 'juxta articular nodules' (see p 2260). The microscopical examination of the contents of the nodules obtained by tapping with a syringe or by excision will clear the diagnosis.

**Prognosis.**—These little tumours are not in any way dangerous to the life or health of the patient.

**Treatment.**—Removal by incision and enucleation is quite easy.

The life history of the parasite is unknown.

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No. 3

### Volvulosis.

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63

## CHAPTER LXXVIII

# DISEASES OF CONNECTIVE TISSUES, MUSCLES BONES AND JOINTS

General remarks Somatic tænia 27—Subcutaneous filariæ—*Dracontia* 3—  
1) retro-conjunctival filariasis—Læsis—Calabar swellings—Dubini's  
filariasis—Myositis purulenta tropica—Gonorrhœa—Boxeraig bones—  
Les gigas—Enlargement of the os calcis—References

### GENERAL REMARKS

THE diseases of the connective tissues which concern us are mostly parasitic being caused by the cysticerci of tapeworms but round worms are also commonly met with especially the guinea worm.

With regard to muscles a disease which must be mentioned is trichinosis which is said to be far from rare in Northern India. Necrosis, caries and tumours of bone are not uncommon but rheumatoid arthritis is rare and tubercular disease at present is very rare. Gonorrhœal and post-dysenteric arthritis are met with and filarial synovitis of the knee joint has been described by Nuttall. Gout is rare but we have seen a typical case in a native who had never left Ceylon.

## DISEASES OF CONNECTIVE TISSUES

### SOMATIC TÆNIASIS

By somatic tæniasis is meant the invasion of the body by the cysticerci of cestode worms. The subject is therefore divisible into—(a) *Cysticercosis* or infection with the larvæ of *Tænia solium* which occurs now and again in the connective tissue of muscles, fasciæ and in the brain (b) *Echinococcosis* which is the infection of the body with the hydrids of *Echinococcus granulosus* and of which we have only met with one example in the tropics and even then it was imported but judging by Begbie's observations it would appear as though the disease was endemic in Ceylon (c) *Spiroganosis* which is the invasion of the body by *Spiroganum* *mussoni*, *S. baxteri* or *S. proflifer* all of which have been sufficiently described in Chapter XXV p 596

may be noticed. In due course a tumour is discovered in the subcutaneous tissue of the chest iliac region. The tumours vary in size from a few millimetres to several centimetres. They may be elastic if they are situated in the fibrous tissue. They usually remain quiescent for years and but seldom ulcerate.

Bernard and Ouzilleau believe that *Onchocerca volvulus* may also give rise to true elephantiasis in certain parts of Africa.

**Diagnosis**—The presence of an elastic tumour somewhat resembling a lipoma or of a firm fibrous tumour in any part of the body of a person who has resided in the endemic region should arouse suspicions as to the presence of *Onchocerca volvulus*.

When the nodules are in the proximity of articulations the condition may closely resemble the juxta-articular nodules (see p 276). The microscopical examination of the contents of the nodules obtained by tapping with a syringe or by excision will clear the diagnosis.

**Prognosis**—These little tumours are not in any way dangerous to the life or health of the patient.

**Treatment**—Removal by incision and enucleation is quite easy.

if the parasite is unknown.

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### Volvulosis

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## CHAPTER LXXVIII

# DISEASES OF CONNECTIVE TISSUES MUSCLES BONES AND JOINTS

osteitis—  
Dubius—  
bones—

### GENERAL REMARKS

THE diseases of the connective tissues which concern us are mostly parasitic being caused by the cysticerci of tapeworms, but round worms are also commonly met with especially the guinea worm.

With regard to muscles a disease which must be mentioned is *trichiniasis* which is said to be far from rare in Northern India. *Necrotic caries and tumours* of bone are not uncommon but *rheumatoid arthritis* is rare and *tubercular disease* at present is very rare. *Gonorrhœal and post-dysenteric arthritis* are met with and *filarial synovitis* of the knee joint has been described by Maitland. *Gout* is rare but we have seen a typical case in a native who had never left Ceylon.

## DISEASES OF CONNECTIVE TISSUES

### SOMATIC TÆNIASIS

it would appear as though the disease was endemic in Ceylon (c) *Sparganiasis* which is the invasion of the body by *Sparganum monsoni*, *S. bixleri* or *S. prolyser* all of which have been sufficiently described in Chapter XXV p 596

### THE SUBCUTANEOUS FILARIASES

**Definition** — The subcutaneous filariases are infestations of the subcutaneous tissue by the adult worms of species belonging to the Filariidae other than *Filaria bancrofti* Cobbold 1877

**Remarks** — The form of filariasis caused by *F. bancrofti* is detailed in Chapter LXVI and now we consider those in which the adult worm lives in the subcutaneous tissue. The varieties of this form of filariasis are dracontiasis or guinea worm infection and dermo-conjunctival filariasis.

#### DRACONTIASIS

**Synonyms** — Dracunculosis Turkish disease

**Definition** — Dracontiasis is the infection of man with *Dracunculus medinensis* (Linnæus 1758) the guinea worm (p. 651)

**History** — The disease has been known from very remote periods and it is probable that the fiery serpents which attacked the Israelites in the desert were guinea worms and that the serpent on the stick was an illustration of the method of extraction advised.

Plutarch (A. D. 50-117) gave an account of the disease as seen on the shores of the Red Sea while Galen (A. D. 131-210) who never saw a case called the disease dracontiasis. Oribasius also mentions it and the worm and Aetius, quoting from Leonides, says that it is found in Ethiopia and India. Paul of Aegina, writing of the upper part of Egypt, there were large tumors formed in the muscular

parts of the body such as the arms, thighs and legs and under the skin in the sides of children which moved and after a time the skin breaks there with a noise like that of a shoe being pulled over a nail. He advises that this be fixed with

it breaks there will be much pain. The nerve and Actuarius writes about its presence in Egypt. Avicenna calls it *Vena medina* after Medina where it was common. He notes the bleb which it makes in the skin and its protrusion after the bleb bursts.

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Egypt Ethiop

common in ne

Cauliac all mentioned the disease and the last named calls the worm *Vena civilis vel medina* while Audry considers it to be an animal. Other writers on this subject are João Rodrigues de Castell Branco (1511-1568), Linscholeri (1599) and De la Motte Lambert (1666). In 1674 Velsch wrote a book on the subject and saw guinea worms everywhere.

The scientific study of the disease and its parasite dates from the work of Fedtschenko in Central Asia in 1870 when he discovered its development in the cyclops, the integument of which he believed the young worms pierced.

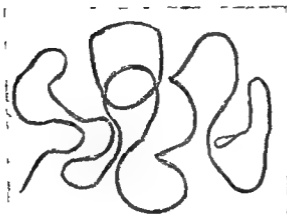


FIG 788A—GUINEA WORM SOMEWHAT SHRIVELLED FROM ACTION OF PRESERVING FLUIDS (HALF NATURAL SIZE)

measuring some 30 inches but during this process she moves about and finally produces the blister where water can be touched and

*Macacus sylvanus*

**Climatology.**—Dracontiasis is a disease of the tropics, especially West Coast. It is also although coolies infected to Ceylon, we have no evidence that the latter island has so far become infected. It is also known in the Fiji Islands. It was introduced into America by the negro slaves, and has become endemic in British Guiana and Brazil.

**Ætiology.**—The causation of the disease is *Dracunculus medinensis* Linnæus, 1758, taken into the body by drinking water containing infected cyclops, which are most abundant during the dry season, and which mostly live near the bottom of wells and collections of water



FIG 789—GUINEA-WORM UNDER THE SKIN

(From a photograph by Christopherson)

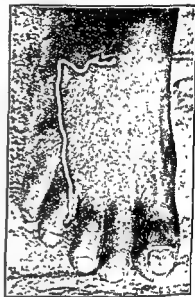


FIG 790—GUINEA-WORM IN PROCESS OF EXTRACTION.

(From a photograph by Christopherson)

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ru-  
the  
on-  
  
ch  
lly  
a vesicle and then an ulcer  
rule produces no symptoms

until a little vesicle appears on the skin or the outline of the worm is noticed under the skin but urticarial eruptions have been observed by several authors and ourselves. The little vesicle bursts leaving a round hollow out of which exudes a clear fluid full of larvæ and at the bottom of which lies the vaginal orifice of the worm. After discharging a certain amount of fluid the anterior

fluid piece of worm is liable to break and if this happens a serious inflammation of the affected area may result. The wound usually quickly heals after the worm has finally been extracted.

there is only one worm but there may be more.

**Blood**—Dudgeon and Child have investigated the blood in this disease and find a marked eosinophilia. The average differential

and the prognosis is good.

The X rays are useful in detecting encysted calcified worms which may cause obscure purulent conditions.

**Treatment**—It is a good plan to massage the area above the vesicle and try with a little patience to get the worm out whole.

worm is either absorbed or tends to emerge and no longer resisting extraction can be easily removed. It has been advised (Emily) to inject swelling not appe

## THE Dermo Conjunctival Filariases

These are Loiasis Calabar swellings and Dubini's filariasis.



## LOIASIS

**Definition**—Loiasis is a subcutaneous and subconjunctival filariasis caused by *Loa loa* (Guyot 1778)

**Remarks**—*Loa loa* which is carried by a species of Chrysops a disco palp over the conjunctiva

eye though accounts of its presence in the vitreous humour require confirmation For description of the worm see p 645

**Climatology**—The geographical distribution of the worm is confined to the West Coast of Africa from Sierra Leone to Benguela but is most particularly Old Calabar the Cameroons and the Ogoon River It penetrates some six hundred miles or more into the interior of Africa

**Symptomatology**—In our experience it usually causes but little disturbance but at times when in the conjunctiva it is associated with piercing and lancinating pains uncertain vision and swelling of the eyelids It is probably the cause of the Calabar swellings mentioned below

**Treatment**—It can be removed by an incision and careful traction Hot fomentations as noted by Elliott cause the worm to come to the surface

## CALABAR SWELLINGS

**Synonyms**—Kamerungeschwülste Tropical swellings Ndito= swelling (Calabar)

**Definition**—Calabar swellings are smooth temporary slightly raised tumours on the head arms hands ankles and feet probably caused by the presence of *Loa loa* (Guyot 1778) and possibly

Cameroc

Ceylon

Ætiol

some way these swellings are caused by *Loa loa* Cobbold 1864 but this has never been definitely proved

definition

that in

## CALABAR SWELLINGS

The way in which the worm induces these peculiar lumps is a matter of conjecture, Argyll Robertson assigning them merely to the movements of the parasite. But this cannot be so otherwise which they can do without any such disturbance.

The next theory is that the parasite irritates the skin, causing the patient to rub the affected area, which in this way becomes mechanically inflamed. This theory was originally propounded by Manson who has relinquished it in favour of another and better explanation.

Two other theories suggest that the parasite, by irritating the nerve endings, either directly or by reflex action causes the swelling, but this does not appear likely, as they ought to occur wherever the worm travels. Manson in 1903 suggested that they might be brought about by the expulsion of the microfilariae from the uterus of the parent worm and this appears not unlikely, and would explain their evanescent character.

Ward is not satisfied with Manson's theory and suggests that they may be due to the expulsion of waste products from the worm. Age, sex and employment have no influence in the causation of the complaint.

**Symptomatology.**—Sometimes without prodromal symptoms or at times after some nausea and headache swellings appear on the head, face arms, wrists hands fingers ankles or toes less commonly on other parts of the body. The reason of this distribution according to some authors, is believed to be the small amount of connective tissue in those regions. The swellings are smooth firm, slightly elevated areas generally about the size of half a goose's egg (5 to 10 centimetres), often painless, though this is not invariable. There is either absence of or only very slight pruritus. They are hot, and do not pit much on pressure. They appear quickly, last for two or three days, and disappear gradually or rapidly and are always associated with an intense eosinophilia. In many cases only one swelling appears at a time.

Stephens gives the differential count of his case as—Polymorphonuclear 26 per cent, lymphocytes, 23 per cent, mononuclears, 1 per cent, eosinophiles, 50 per cent.

**Diagnosis.**—There is no difficulty in recognizing these fugitive swellings in persons who have resided in the endemic region. The presence of extremely well-marked eosinophilia may help in the diagnosis.

**Prognosis.**—They have never been known to cause serious symptoms, but may recur for many years after the patient has left the tropics.

**Treatment.**—This is unsatisfactory but cool applications such as diluted liquor plumbi (5 per cent) may be made to the swellings and an ichthyol ointment or lotion applied.

**Prophylaxis.**—As our knowledge of the life cycle of *Loa loa* is not possible to lay down rules for the prophylaxis.

## DUBINI'S FILARIASIS

**Definition**—Dubini's filariasis is a dermo-conjunctival filariasis caused by *Filaria conjunctivæ* Addario 1885

**History**—The immature female worm was originally discovered by Dubini in the sut  
possibly the same a  
von Nordmann 183  
nee Wilson 1844

worm in a calcified nodule in the gastro-splenic omentum of a woman in Budapest. He named it *Filaria peritonei hominis*. In 1895 Addario named a female worm extracted by Vadela from the conjunctiva *F. conjunctivæ*. Vadela's case was a woman from Catania in Sicily. In 1887 Grassi gave a full description of the female worm calling it *F. inermis* because of the absence of papillæ on the head. In 1906 Alessandrini found it in an abscess in subcutaneous tissue of the arm and in 1918 Graham Forbes met with two cases in the subcutaneous tissue of the forearm and of the nose and was

## Francaviglia

**Climatology**—The worm is found in Italy, Sicily, Hungary, Macedonia and Roumania. It is a parasite of the horse and ass and but rarely of man.

**Ætiology**—It has been suggested that the worm is introduced by the bites of *Chrysops excrucians*.

**Morbid Anatomy**—The nodule containing the worm consists of fibrous connective tissue with round-celled infiltration and traversed by lymph spaces.

**Pathology**—It is thought that the worm enters a lymphatic canal which becomes cut off and is surrounded by an inflammatory reaction. No microfilaræ can be found in the blood or in the nodules.

**Symptomatology**—Marks of a bite have been seen in only one case. Usually it is a small tumour in some area of the body which is the first sign to attract attention.

The differential blood count is as follows—

	Per Cent
Polymorphonuclear leucocytes	47.0
Mononuclear leucocytes	10.5
Small lymphocytes	38.0
Eosinophile leucocytes	3.5
Basophile leucocytes	1.0

Some hot and swollen  
symptoms have lasted  
over to recur again in

about ten to fourteen days and this cycle recurs and recurs. Some-

### DISEASES OF MUSCLES

times an abscess forms in which the worm or its remains can be found  
Treatment —The correct treatment is to excise the nodule  
Prophylaxis —Nothing can be said under this heading as the method of infection is unknown.

### DISEASES OF MUSCLES

#### MYOSITIS PURULENTA TROPICA

Synonyms —Mumi fever (Samoa) Bungpaggia (Northern Gold Coast)

History —This condition has been observed in various parts of the tropics including the northern territories of the Gold Coast by several observers among whom Van Polk, Zemann, Kütz may be mentioned. The last named author has given a good general account of the malady.

Chromatology —The disease is found in tropical Africa and Samoa.

Aetiology —Some authorities suggest that it is due to a *Filaria* in the limbs reported will fever of a remittent or intermittent type. Abscesses form in the muscles in various parts of the body.

Treatment —This is surgical the abscesses being evacuated.

### DISEASES OF BONES

#### GROUNDON

Synonyms —Anákhre=big nose Henpuye=dog nose  
Definition —Groundon is a disease of unknown causation characterized by a bony swelling usually bilaterally symmetrical situated on either side of the nose.

History —The disease was first described by Macalister in 1864 under the term of the horned men of Africa. Lamprey also referred to it in a West Indian child in 1864. Macalister described the disease which he saw on the Niger under the name of groundon or anákhre. In 1900 Chalmers gave an account of the disease as seen on the Gold Coast and Renner as seen in Leone. Later Braddon recorded a case in Malaya and found the disease in Sumatra and in China. Friedrich an excellent description of it as seen in East Africa and an account of his cases on the Gold Coast while Cantlie described the disease in a European. Lastly Roubaud and Bouffard and more recently Blum and Blum have recorded cases of unilateral groundon in a European. Lastly Roubaud and Léger have recorded cases in monkeys.—P. The first three observers found that the condition affected the bones of the skeleton besides the nasal bones. Letullier and Callitrichous found that the condition affected the bones of the skeleton in the skull of an ancient



**Symptomatology**—Generally the disease begins with pain in the

and steadily these lumps increase in size increasing with time (it is said at times destroying the eyes) and giving rise to a hideous deformity rather like a *Cynocephalus* monkey and hence called dog nose. When fairly well developed an oval bony swelling with its long axis directed downwards and outwards is seen sym-

with the disease

**Varieties**—Instead of being bilaterally symmetrical the bony lump may develop only on one side of the nose. Orpen has described in addition to the two usual tumours a third in the malar region.

merely easy but most effective as the disease is known not to have returned some six or seven years after the operation.

**Prophylaxis**—As the causation is doubtful nothing can be said under this heading.

### BOOMERANG BONES

**Synonym**—Boomerang leg

**Definition**—A disease of the long bones commencing gradually and associated with pain tenderness and longitudinal bowing of the bones which remain permanently deformed after the acute symptoms have disappeared. Several conditions are apparently

**Climatology.**—The disease is known in the northern portion of Australia the islands of the Torres Straits in the Sudan in British New Guinea

**Ætiology.**—This is unknown It is not syphilitic tubercular osteomalacic nor due to rickets Black considers the condition to

**Morbid Anatomy.**—The bending in the tibia takes place at the junction of the upper with the middle thirds The bone is heavy and compact in the centre and freer at the ends The narrow cavity is almost filled in with compact bone



FIG 792.—BOOMERANG DISEASE  
(From a photograph by Christopherson)

the children start to walk and the bones bend forward gradually thus giving rise to the characteristic deformity, which becomes permanently fixed

### PES GIGAS

**Synonym**—Congenital partial hypertrophy of the foot

“ home for  
a young  
rand in a

European.

**Symptomatology.**—The condition is congenital, and may be studied by the X rays when it will be seen that the three inner meta-

tarsal bones are hypertrophied and the second and third united while the terminations of the toes are club like. In Cousland's case there was marked hypertrophy of the calf muscles.

**Treatment**—It has not been treated so far as we know.

### ENDEMIC ENLARGEMENT OF THE OS CALCIS.

**Definition.**—Endemic enlargement of the os calcis is a disease of

recurrences take place then

**Symptomatology**—The disease begins suddenly with fever, great tenderness over the os calcis with local pruritus so severe as to prevent sleeping and walking. In three to seven days the outer surface of the bone begins to increase in size and continues to do so for some two weeks during which time the pain lessens and the temperature falls to normal.

In about a month the swelling reaches its maximum size at

outer surface as a rule but may also attack the posterior third of the bone.

Rarely it attacks other tarsal bones but has never been reported as affecting a joint. Yearly recurrences during the wet season are common.

**Treatment**—Medical treatment is useless but relief is given by cutting down on to the affected area and trephining a hole into the bone.

**Prophylaxis**—Concerning this nothing is known.

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## CHAPTER LXXIX

### DISEASES OF THE NERVOUS SYSTEM

General remarks—Endemic paralytic vertigo—Latah—Bangs—Schamanism—Amok—Tropical neurasthenia—Idiomatic peripheral neuritis—Erythromelalgia tropica—Desert hallucinations—Emotional excitement—Delusions—Auto suggestion—References

#### GENERAL REMARKS

THE nerve diseases of the tropics include many general diseases already dealt with—e.g. pellagra, leprosy, beriberi, etc. Put apart from these conditions, *meningitis cerebralis*, *neuritis cerebralis*, rarely, *general* though the most commonly met with are endotheliomata and tubercular or syphilitic lesions. *Hysteria* is common among the better class natives especially among the young women educated according to Western ideas.

neurasthenia, endemic vertigo and other conditions must be briefly touched upon.

*Har Zotte Negroes*—The recent war has greatly improved the

and shell shock.

The pathology of some of these conditions has been carefully investigated by Mott. Many persons suffering from shell shock have been permitted to go on duty to the tropics with often unpleasant mental results. These persons are especially liable to be influenced by the sun's rays.

### ENDEMIC PARALYTIC VERTIGO

The vertigo Ptosique Gerlier's vertigo is characterized by eye symptoms such as ptosis, dimness of vision and by paralysis of the neck and extremities.

**History**—The disease was first described in 1884 by Gerlier in Ferney in Switzerland in 1886 and by Miura in 1894 in Japan.

**Climatology**—It is known in France, Switzerland and Japan where it is found in the northern provinces and in the island of Shikoku. It begins in spring, attains its greatest numbers in summer and ends in autumn.

**Ætiology**—The ætiology is entirely unknown and hence there are many theories which suggest alcohol, poisoned bread and lentils and latent malaria, but the one which is favoured by both Gerlier and Miura is the association of the disease with stables. Gerlier says that in the valley of the Lake of Geneva it is not unusual for people to sleep in the stables and Miura says that in the regions affected in Japan it is usual to have the house so arranged that a part is used as a stable while the remainder is occupied by the family.

Further it is associated with warm weather. Miura gives as

**Symptomatology**—An attack begins with a blurring of objects, everything appearing as if in a fog together with ptosis and less

of power in mastication and in bad cases of deglutition. At the same time paralysis of the muscles of the back of the neck, the back and the extremities are observed. The head falls forward and is only raised with difficulty hence the name of the disease.

kubisagari which means he who hangs his head. If the muscles of the back are paralyzed there may be difficulty in raising the

be a little ptosis or weakness of the muscles of the back neck hands or legs and increased reflexes

**Diagnosis**—The characteristic symptoms are Ptosis and the falling forward of the head and neck. These symptoms differentiate it from the other forms of vertigo

**Prognosis**—The disease is never fatal though it may last for many years

**Treatment**—The first requirement is removal from the endemic area and from living in close proximity to stables. Medicines are not of much use but a combination of potassium iodide and arsenic is recommended

### LĀTAH

ong Bah

gestion is  
echolalia  
t always

conscious

**History**—The convulsive tics have been carefully studied in Europe by Charcot Gilles de la Tourette and Guinon but the allied condition lĀtah found in Malaysia as well as similar conditions found in

investigated  
in 1892 Van Br  
in 1907 Fletch

a fair amount of definite information with regard to lĀtah is available

**Climatology**—LĀtah is found principally in the Malay Peninsula Java Sumatra and is also known in Siam in Burma in the Philippines Siberia and among the Jumpers of North America. Fletcher has rightly drawn attention to the fact that while the disease is very common in the Malay States it is apparently rare in the Malays who have emigrated. Thus for example it does not occur among the large colony of Malays in Ceylon which may be due to the fact that they originally came from Batavia in Java

**Etiology**—The exciting cause appears to be any sudden start

producing some peculiar movement after which any unlooked for action may be imitated and is generally accompanied by bad language. The mildest form of the disease is merely an exclamation or a scream when startled but in severe cases the patient will imitate any sudden motion or obey any suggestion made to him.

Fletcher relates that in some parts of the Malay States it is occasionally impossible for a judge to examine the witnesses as they can do nothing but imitate and repeat the questions put to them.

According to Abraham the exciting causes are (1) Auditory—*e.g.* an unexpected noise behind the person (2) visual—some un

seeing or  
touch the  
ed and at  
the same time may use foul language. The unfortunate victim

<sup>u a m</sup> It was about this time that a number of other people in my household began to develop signs of the affliction. I must not be understood as suggesting that



Fl aspect of látah  
 su everely affected  
 látah subject to  
 He concludes that in látah

second person

a near connection between látah and crime

**Treatment.**—The treatment is most unsatisfactory, but auto-suggestion might be tried. Abraham states that some patients have cured themselves by determination not to succumb.

### BANGA.

**Definition.**—A hysterical condition chiefly influencing women, but also occurring in men in the Welle. It is characterized by a sudden onset, and is induced by an attack, in

### SCHAMANISMUS.



## AMOK

**Definition**—Amok is a psychical disturbance which after a period of depression suddenly develops into a violent attempt to kill people of which no memory may be left and after which a stuporous condition supervenes.

**Remarks**—Amok which means an impulse to murder is a disease frequently found among the Malays but also occasionally in other Oriental races Abraham thinks that there may be some relationship between amok and latah

**Climatology**—Amok is found in Malaysia among the Bugis of Celebes the Malays of Indo-China Malacca and the Malay States It is also said to occur at times in Trinidad and among the inhabitants of India and Siberia

**Ætiology**—The exciting cause appears to be a strong emotion of anger sorrow or fear after which a pause or incubation period of depression follows which may last for days or weeks during which the patient broods over his wrongs The Dutch believe that it is induced by opium smoking and Muall that it is due to smoking haschisch (*Cannabis indica*) but these causes are insufficient

**Symptomatology**.—The attack is ushered in by colour sensations of red or black with or without vertigo and the patient complains of the appearance of devils which he attempts to kill The amoker usually rushes out of his abode and attacks friends or foes  
s kris or flame-shaped knife  
the attack usually lasts but a  
during that period are not

stage as far as we know

## TROPICAL NEURASTHENIA

in Americans returning from the Philippine Islands

**Ætiology**—Tropical neurasthenia is apt to develop in white people in any part of the tropics but especially in those which are

ful  
not  
ad  
od  
les



which is 5 000 to 6 000 feet high and is situated between 13° to 14° south latitude and is inhabited by the Bihe Bailundo and

maize or nerve-starvation but he is not satisfied with any of

revealed nothing and post mortem the nerves were found normal

damp and diminished by heat and dryness . They may also appear  
any part of or all over  
appearing to walk on  
for years but spon

taneous recovery is known

**Diagnosis**—The diagnosis is easy for it is distinguished from beri beri by the absence of pain in the calves by the absence of the heart symptoms and of the paralysis from elephantiasis by the swelling being transitory from malaria by an examination of the blood and from pellagra by the symptoms improving during the warm season

**Prognosis**—This is good as to life and general health though the course of the malady may be very protracted

**Treatment**—No method of treatment is known to do any good.

### MYTHROMELALGIA TROPICA

Under this term Gerrard describes a nerve condition in which natives who work barefoot on roads and plantations in Malaya complain of a feeling of pins and needles in the soles of the feet  
and  
ent  
to

### DESERT HALLUCINATIONS

**Synonym**—Le Ragle

D Escayrac de Lauture describes hallucinations of the senses of sight hearing smell or taste or even of common sensations which  
The causation  
night but  
be more



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# CHAPTER XC DISEASES OF THE ORGANS OF SPECIAL SENSE

General remarks—Diseases of the eye—Diseases of the ear—Diseases of the nose—References

## GENERAL REMARKS

It is not usual to consider the diseases of the organs of special sense in a work on tropical medicine but all the diseases of the tropics are being carefully studied at the present time and therefore we are of the opinion that a few cursory remarks on the above subjects from the point of view of the general practitioner may be of some interest

## DISEASES OF THE EYE

The history of the study of eye disease in the tropics has still to be written but anyone interested in the general history of this branch of medical science is referred to Hirsch's *Geschichte der Augenheilkunde* (Leipzig 1877) which though issued as a separate little volume was originally part of Graefe's and Sæmisch's *Hand buch der Augenheilkunde* vol vii. In this book there is an account of the ophthalmology known in ancient Egypt and India as well as the history before and during the Alexandrine period of medical development. It will suffice to say that eye disease is described and treated in the Ebers papyrus and that Susruta mentioned 76 diseases of the eye of which 9 were of the black part of the eye 21 of the eyelids 11 of the sclerotic 4 of the black part and 2 were injuries. In recent years the work of the ophthalmic surgeons of India and Egypt and elsewhere has been beneficial not merely in an extension of knowledge but more importantly in benefit to the communities of the lands in which they live. Excellent work has been done by private generosity in Egypt which goes such as those provided by the travelling ophthalmic hospitals from district to district. By this means natives at a distance from which can be carried on in the interval between a visit by the local hospital and dispensary. Another excellent institution of more or less recent origin is the Central Ophthalmic Hospital to which the patient can be

sent or more prolonged expert treatment. A model hospital of this nature can be found in the Victoria Eye Memorial in Colombo Ceylon. The prevalence and importance of eye disease in the tropics cannot be too strongly insisted upon. The cases of oph

to the presence of large numbers of flies at certain seasons which also materially assist in the spread of infection from the diseased to the healthy. Moreover these factors are reinforced by the irritation caused by the glare of the sun and by the dust. Blindness is also very prevalent in the tropics. Denham in his report on the census in Ceylon in 1911 states that in a population of 4 106 350 persons there were 3 957 blind persons which figures when analyzed showed that there were 11 blind men and 8 blind women in every 10 000 men and women in Ceylon. In India in 1901 there were 12 blind men and 12 blind women per 10 000 of each sex while in England in 1901 there were 8 blind men and 7 blind women per 10 000 of each sex.

Dr Andreas Nell in his statistics of the Victoria Eye Memorial Hospital in

113

114

2

**Constitutional Diseases**—These include syphilis malignant tumours leprosy tuberculosis and enteric fever but malarial cachexia was not met with as a cause of blindness nor was ankylostomiasis.

**Drugs**—Alcohol (most common) tobacco (rare) quinine and opium (very rare).

There can be no doubt that blindness arises from neglect of the trivial cases of eye disease and from septic infection and that

## A EYE DISEASES PROPER

### Edema of the Eyelids

Under this term Chalmers and Marshall mention the acute swelling of the eyelids in Europeans and natives in Khartoum. The

## EYE DISEASES PROPER

ected area shows the mark of a bite and may perhaps be due to  
ant perhaps of the genus *Monomorium* Mayr 1855

### Congenital Defects

These are by no means uncommon in the tropics and our experi-  
ence includes cases of apparent anophthalmus or microphthalmus  
coloboma and albinism

### Colour Blindness

This is of importance and natives who are to be employed as pilots  
or on railways should be examined as to their power of distinguishing  
colours and indeed if this has been neglected it is advisable to  
examine the existing employes as the results of such examinations  
are sometimes surprising In testing only the Eldridge-Green  
lamp and method should be used.

### Errors of Refraction

A subject which has begun to attract a considerable amount of  
attention is the condition of the eyes of native children in the more  
modern and higher class native schools as regards errors of refrac-  
tion but the medical inspection of native schools in the tropics is  
at present neglected though the children suffer from what may be  
termed book hunger and so strain their eyes excessively

### Foreign Bodies

In addition to the usual foreign bodies met with in the Temperate  
Zone small flies are apt to get into the eye in the early evening  
and some of these are very irritating and may cause congestion  
r by introducing micro-organisms conjunctivitis Chalmers and  
Marshall record the finding of a small ant *Monomorium bicolor* var  
*nitiventre* firmly fixed by its jaws on to the ocular conjunctiva in  
a person in Khartoum

### Pterygium

In this disease the pinguicula being irritated spreads on to the  
corner and carries th conjunctiva with it thus giving rise to  
triangular folds extending from the ocular conjunctiva to the cornea  
on the inner or outer aspects of the eye It is very common in the  
tropics especially among coolies in Indo-China  
The treatment is removal

### Hypertæmia of the Conjunctiva

This is extremely common being caused by the glare of the sun  
or by the dust especially in sandy regions and is especially a  
error of refraction. It is also more liable to occur in persons who  
have some congestion of the naso-pharynx and is one of the sym-  
toms of rhinitis spastica visomotora Usually the symptoms  
but slight but the eyes are inclined to water this is aggrav



by exposure to light while the patient may complain of a slight feeling of grit in the eye or of a burning sensation.

On examination a portion of the palpebral and of the ocular conjunctiva is seen to be congested while the secretion from the Meibomian glands is noticed to be increased in amount and the eyes to be watery. Treatment consists in bathing with warm 2 per cent solution of boric acid in giving the eye rest and in wearing neutral tinted or yellowish tinted glasses (Xantl ophylline) which can be obtained in three strengths—light medium or strong

### Conjunctivitis

This is extremely common in the tropics among natives and Europeans and may be clinically subdivided into—(1) Conjunctivitis catarrhalis (2) Conjunctivitis gonorrhoeica (3) Ophthalmia neonatorum (4) Conjunctivitis trachomatosa (5) Epithel

but can be caused by caterpillar hairs or plant hairs becoming embedded in the conjunctiva

### Conjunctivitis Catarrhalis

This may be subdivided into the acute the chronic and the follicular forms.

**The Acute Variety**—This is very common in the tropics where it may occur in epidemics. The most common cause is the Koch-Weeks bacillus which may induce a very severe form of inflammation while almost as frequently the Morax Axenfeld bacillus causes a mild conjunctivitis lococcus.

may disappear in eight to fourteen days if untreated but more usually it becomes chronic. The complications most commonly observed are corneal ulcer and iritis

The best treatment in severe cases is to lightly mop the inflamed eyelids and to silver nitrate followed by solution of instilling a

solution

to prevent the sticking together of the eyelids a 2 per cent of boric acid or a  $\frac{1}{2}$  per cent strength of white precipitate may be used

It is unnecessary to state that no bandage should be

certain that trachoma is absent. In the acute form the treatment is the same as for acute catarrhal conjunctivitis but in the chronic it is usual to apply copper sulphate treatment. An ointment of 1 in 1000 copper sulphate or 1 in 100 copper citrate is recommended by some authorities.

#### **Conjunctivitis Gonorrhoeica**

It is well known that no special reference need be made

#### **Conjunctivitis Neonatorum**

is usually due to the gonococcus but may be caused by a virus or probably by a ciliated protozoan

## Micrococci and Conjunctivitis.

The presence of *Cocc...*

Organism	Glucose	Maltose
Gonococcus	+	-
Meningococcus	+	+
Micrococcus catarrhalis	-	-
	Acid only +	acid and gas +
	neither -	

## Conjunctivitis Trachomatosa

Synonym *Ocular...*

chi  
in  
S-

ent of the  
so common  
Japan and

fungus—*eg* th...  
by Noiszewski  
by the smallest  
it occurs in the  
increase in size  
from granules in which subsequently very minute granules appear

Probably *Trich...*

been found in the vaginal discharge of women whose children have suffered from the form of ophthalmia neonatorum in which no gonococci or streptococci can be found. Further Castellani has found similar bodies in a case which may have been one of these.

resembling the conjunctivitis trachomatosa of man

**Method of Infection**—The infection is carried by the hands, towels, handkerchiefs etc., from the sick to the healthy. There is no evidence of aerial transmission. The agency of flies in the transmission of eye disease has long been known—for example Budd in 1862 considered it proven that they transmitted ophthalmia.

There were few Nuttall and Jephson consider the spread of

Another factor may be some local derangement as for example a  
slight attack of conjunctivitis or some general derangement as  
for example an attack of fever

**Pathology.**—The essential feature of the disease is a round celled infiltration  
of the conjunctiva associated with hypertrophy of the papillae of that  
membrane

Microscopic description of vessels

Conjunctiva is studded with the typical nodules but these may not  
be apparent and may even at first be absent. These acute attacks

**I. T.**  
The diff  
and it i  
hypertro  
) exar  
The granules are not so prominent and are there  
fore less easily seen. A trachoma granule is typically a grey  
reddish translucent granule comparable to a grain of boiled sago  
in appearance. Harston's sign is a linear groove running almost

horizontally outwards from the external canthus. The hypertrophy of the conjunctiva increases until some indefinite limit is reached when it ceases and cicatrization sets in but if the hypertrophy is excessive it may last for years and the cicatrization may develop slowly but surely.

The cicatrization shows itself at first as whitish striæ on the tarsal conjunctivæ which becoming more numerous unite into a net work the meshes of which are occupied by the hypertrophied conjunctiva which gradually diminish as cicatrization proceeds until the conjunctiva becomes pale in colour. In some cases this cicatrization is very slight and hardly noticeable while in others it may produce serious complications.

The loss of vision is due to pannus and ulceration of the cornea the former being a deposit of vesicular gelatinous tissue on the cornea which becomes uneven and raised in fine projections while the latter may occur with the pannus or separately.

The sequelæ of trachoma may be classified into —

(a) Corneal ulceration and pannus leading to opacities  
 (b) Cicatrization leading to (1) *Trichiasis* in which the cilia are turned backwards and may touch the cornea (2) *Entropion* in which the border of the lid is turned backward (3) *Ectropion* in which the lid is everted (4) *Syrblepharon posterius* in which the fornix is diminished in depth and the lid is tightly fastened to the eyeball.

(c) Conjunctival xerosis in which the conjunctiva owing to atrophy loses its secreting powers and becomes dry and shrunken.

**Treatment** — The acute cases are to be treated with silver preparations as described for conjunctivitis.

When the follicles are well developed it is usual to express them by means of a flat Grady's forceps or by the roller forceps of Knapp but this must be performed under an anæsthetic. The ruptured surface is then punted with a solution of perchloride of mercury and after one or two days interval the daily application of solid

ure or as a  
 0.05 gramme  
 mine gum  
 ured. The  
 centimetres

in length

Harston strongly recommends treatment by carbon dioxide snow

by cocaine which therefore need not be used.

Hegner and Baumm have advised treatment by quartz light while other methods are Galezowski's excision of the retrotarsal folds, Kuhnt's removal of the tarsus while treatment by Merl's extract of abrin, Mayon's X ray method and Treacher Collins's radium treatment may be mentioned.

## Epitheliosis Desquamativa.

causation to be a chlamydozoon—*Lycomon atrophicans* Leber and von Prowazek, 1911—which is found in the milky secretion in the

but atrophy of the conjunctiva ensues. The treatment recommended is pyoktann in the proportion of 1 in 1 000 to 1 in 100.

## Conjunctivitis phlyctenulosa

This is not very frequently met with in the tropics. It is usually found in acrofulous individuals and is characterized by the presence of small vesicles each surrounded by a red-lined zone.

## Conjunctivitis Vernalis

**Synonyms**—Conjunctivitis Æstivalis Spring catarrh

**Definition**—A chronic form of conjunctivitis resembling conjunctivitis trachomatosa but occurring in the spring and summer.

**Ætiology**.—The cause is unknown but some authorities consider that it is due to sunlight while others hold that it is an infection from the skin.

**Symptomatology**.—On the approach of the warm weather in spring the eyes begin to itch and red and photophobia is experienced. The conjunctiva is seen to be covered with a bluish white film which in summer waxes in the autumn and disappears in the winter only to recur in the spring.

**Diagnosis**.—The diagnosis must be made by the history and by the bluish white sheen over the papillæ. It resembles trachoma but

the granules are broader harder and paler while the history is distinctive

**Treatment**—Protective glasses must be worn and zinc sulphate lotion ( $\frac{1}{2}$  per cent) dropped into the eyes. If the itching is very severe it is recommended to apply a few drops of a very weak solution of acidum aceticum dilutum. Iron tonics are also advised.

### Epithelial Xerosis of the Eye

This condition has been recorded several times from the tropics.

Archibald has recently described very carefully three cases in the Sudan. The xerotic patches were situated on the conjunctiva external and close to the corneal margin of both eyes (Fig 793). The patches were greyish white in colour and of a soft viscid consistency the superficial layers were readily removed with a platinum loop the deeper layers however were more adherent to the subjacent epithelium which showed a brownish pigmentation. The



FIG 793—EPITHELIAL XEROSIS OF THE EYE

(From a photograph by Archibald)

bloodvessels in the vicinity of the patches were congested. There was no evidence of trachoma or inflammation of the eyelids. The whole bulbar conjunctiva was moist and could be readily moved into folds by gentle pressure with a platinum loop. There was an absence of pain or discomfort associated with the lesions which appear to have had a duration of several years. Vision was not impaired and there was no evidence of night blindness.

**Ætiology**—Scrapings from the xerotic patches showed the

numbers which was obtained in organism (*B. xerosis* Neisser) differed from the true Klebs Loettler bacillus in its cultural reactions and moreover was non pathogenic

health and the administration

The affected eyes should be treated with weak boric solution drops of a 1 per cent solution

of protargol

### Lachrymal Sac

The lachrymal sac being in direct connection with the nose is very liable to harbour micro-organisms and fungi and is therefore a possible source of infection for the conjunctiva and cornea

## Cornea

Phlyctenular keratitis is common as a sequela to conjunctivitis

## Ulcus Serpens Corneae

This appears as a greyish white or yellowish slightly depressed disc near the centre of the cornea the rest of which is clouded. It is associated with iritis and turbidity of the vitreous hypopyon and more or less œdema of the lids with conjunctivitis cyclitic photophobia and pain except in the so-called torpid cases. The ulcers increase in depth and extent perforation occurs into the anterior chamber which contains sterile pus which now escapes and at the same time prolapse of the iris may occur. The inflammation now comes to an end but panophthalmia may occur. The most

ment followed by application of atropine iodoform or orthoform powder or 1 per cent xeroform ointment or calvaryol ointment

are best treated by iridectomy on each side of the synchia. Harston operates on the worst eye of the two when both are involved as operative interference is apt to set up a severe irido-cyclitis in the poorly nourished Chinese coohe with eventual loss of sight in the eye. The nasal infection must also be treated by antiseptic douches.

## Iris

Diseases of the iris are as common in the tropics as in the Temperate Zone. Those which are specially connected with tropical disease will be mentioned below but it is well to remember that in native races the stroma of the iris is laden with pigment which probably accounts for its slow reaction to mydriatics.

## Cataract

operation for intracapsular extraction

Sometimes in the tropics it is impossible to wait until the cataract ripens and operations may have to be performed on immature cataracts. To meet this difficulty Nell has devised a modification of the usual procedure which consists in performing a zonulotomy



by means of a special hook introduced through the corneal wound made for a cataract extraction and under the iris. After this preliminary proceeding he completes the operation by intracapsular extraction of the lens.

### Glaucoma

Acute glaucoma is rare in the tropics while chronic glaucoma is common but the signs symptoms and treatment are the same as in the Temperate Zone. The Elliot operation of trephining with or without subsequent iridectomy is recommended.

### Fundus Oculi

The colour of the fundus oculi depends upon the amount of retinal pigment present and to a less degree upon the amount of the choroidal pigment visible. It therefore shows variations from the European standard in dark and yellow races. In these races the amount of retinal pigment is increased and may completely conceal that of the choroid and so produce a uniform yellowish colour as seen in the Chinese or a dark reddish or even an almost greyish-

### Sunlight

The effect of sunlight on the eyes has been studied by Sisson who considers that there is ample proof that light injures the eye and that it may possibly be the cause of some diseases of the eye.

Sisson considers  
excessive light  
injurious

## B EYE COMPLICATIONS OF TROPICAL DISEASES

### Malaria

Malaria is held to be responsible for conjunctivitis, serpiginous corneal ulcers, malarial iritis and retino-choroiditis as well as amaurosis.

Until a few years ago the majority of the fevers of the tropics including enteric fever were classified as malaria and any local affection of an unknown nature was also classified in the same manner. The case of conjunctivitis in which one of us found cell inclusions would a few years ago have been named malarial. We are therefore of the opinion that the existence of malarial conjunctivitis has not been proved.

**Keratitis Dendritica**—This keratitis is that variety of herpes corneæ febrilis (sometimes called Herpes corneæ zoster) which gives rise to those ulcers (formed from the ruptured herpetic vesicles) which extend in certain directions only as grey forked striæ with lateral branches which break down thus forming a

branched ulcer with grey margins. The ulcer then becomes clean and heals after one to three months leaving a branched opacity of the cornea. This is one form of corneal ulcer to which the name serpiginous is given but it equally applies to *ulcus rodens* and keratitis marginalis superficialis which as far as we know have never been stated to be of malarial origin. Keratitis dendritica was first described by Kipp in America who stated that nine out of every ten cases were malarial.

The treatment must be that described for malaria as well as constitutional and it is usual to recommend that the ulcers be treated by a piece of blue stone whittled to a fine point and carried carefully along the furrow made by the ulcer or by careful cauterization by the actual cautery.

**Keratitis Profunda**—Synonyms—Keratitis parenchymatosa circumscripta or Central parenchymatous infiltration of the cornea.

This is said by Arlt among other causes to be brought about by chronic malarial cachexia. It begins with a grey opacity situated in the middle and deep layers of the centre of the cornea and over which the corneal surface is grey and punctate. Seen with magnifying glass the opacity resolves into dots and maculae or grey interlacing striæ. After remaining four to eight weeks it slowly abates without ulceration. The iris may be hyperæmic and the eye may or may not be symptomatic of inflammation of the cornea which would be the same as for malaria but it must be definitely stated that the cause is unknown.

**Iritis**—Secondary iritis is said to be very rarely caused by malaria but even this is open to doubt.

**Vitreous Opacities**—Hæmorrhage and serous effusion have been described.

**Amaurosis**—Malarial amaurosis is described in Chapter p. 118r under the term Amaurotic Pernicious Fever but occur not merely during an attack of malaria but as a sequel and as such was described by Jacobi in 1868 and later by Chalmers. It is due to atrophy of the optic nerve.

**Retino-choroiditis**—This was first described by Poucet as occurring in chronic malaria but it is also found in the acute and chronic forms. In the acute affection the vessels of the retina are filled with corpuscles many of which contain malarial parasites. In the choroid the larger vessels contain pigmented leucocytes of which contain red corpuscles with malarial parasites. The retina is hazy the papilla is obscured and there are retinal hæmorrhages. The amblyopia may be temporary or permanent. In the chronic condition there is atrophy of the capillaries of the choroid. The macular region shows small whitish dots in the centre of which retinal pigment can be seen. These changes extend to the periphery. Pathologically they are colloid masses in Bruch's membrane.

**Suppurative Uveitis**—Suppurative choroiditis leading to blindness has been described by Pennoff.

**Accommodation Paralysis**—Accommodation paralysis and spasm of the muscles of accommodation have been described by Bull and Slitting

### Trypanosomiasis

Choroiditis cyclitis iritis and optic neuritis of a temporary nature are reported as occurring in sleeping sickness as well as engorgement of the iris and loss of the light reflex with wide dilatation of the pupil

### Relapsing Fevers

Irido cyclitis or iritis is a frequent complication of the relapsing fevers and though it ultimately ends in a cure, it is usually protracted

### Plague

— 1 ter  
a all  
fo la  
tion of atropine and blood extraction by leeches

### Leprosy

In tubercular leprosy yellowish translucent nodules of a non vascular nature may develop in the conjunctiva near the cornea Iritis and cyclitis with or without the formation of nodules may occur According to Wood more than half the anæsthetic lepers of South Africa suffer from eye complications—e.g. paralysis of the orbicularis palpebrarum ectropion of the lower lids epiphora, corneal opacity and ulcerations Of the tubercular lepers he says 90 per cent are affected in the first ten years of the disease and if they survive they become blind They suffer from invasion of the lids conjunctivæ sclerotic cornea and iris by the disease and in addition may suffer from paralysis of the ciliary muscle and irido-cyclitis Heymans finds lymphophthalmos to be common In 1915 Stanziiale published experiments on the eye of rabbits injecting leprotic material into the cornea

### Cholera

Focal necrosis in the cornea and conjunctiva has been reported in convalescence from cholera while a form of cataract depending upon the abstraction of water takes place at times in the last stage of the disease

### Dysentery

Conjunctivitis keratitis iritis and irido cyclitis are reported in dysentery both amœbic and bacterial

### Hikan

Under this term a curious disease is described by various observers in India and Persia The main symptoms are  
the skin and  
and Iran

**Quinine.**

Amaurosis associated with complete deafness, may set in after

and do not react to light, while in the latter they do react to light

**Atoxyl.**

Sudden amaurosis may be caused by atoxyl

**Animal Parasites.**

**Ocular Filariasis**—*Loa loa* has been noticed in the ocular and

## Ophthalmomycoses.

*Glenospora graphis* and Liegard and Landrieu a form of conjunctivitis due to *Nocardia dassonvillei* while in 1912 Landrieu reviewed the whole subject of ocular mycoses in a singularly able manner

Fungi.—The following fungi have been recognized in various diseases of the eye —

## A. ORDER ASCOMYCETES

## I Suborder Gymnoascees

## (a) Family Saccharomycetes

(1) Genus *Cryptococcus*

*C. dermatitidis* Gilchrist and Stokes 1898

(2) Genus *Saccharomyces* several species

## (b) Family Gymnoasceæ

(1) Genus *Microsporon*

*M. lanosum* Sabouraud 1907

(2) Genus *Trichophyton*

*T. tonsurans* Malmsten 1845 and other species of the same genus

(3) Genus *Achorion*

*A. schoenleinii* Lebert 1845

## II Suborder Carpoascees

## Family Perisporiaceæ

Genus *Aspergillus*

*A. fumigatus* Fresenius 1775

## B. ORDER HYPHOMYCETES

(1) Genus *Nocardia*

*N. bovis* Harz 1877

*N. israeli* Kruse 1896

*N. foersteri* Cohn 1874

*N. dassonvillei* Brocq Rousseau 1907

(2) Genus *Monilia*

*M. aidicans* Robus 1853 and other species of the same genus

(3) Genus *Glenospora*

*G. graphis* Siebenmann 1889

(4) Genus *Sporotrichum*

*S. beurmannii* Matruchot and Ramond 1905

Pathogenicity.—The ocular diseases caused by the above fungi may be grouped as follows —

## Fungus

## Disease

Cryptococcus dermatitis and various species of the genus Saccharomyces	Ocular Blastomycosis
Microsporion lanosum	} Ocular Trineæ
Trichophyton tonsurans	
Achorion schoenicini	
Aspergillus fumigatus	Ocular Aspergillosis
Nocardia bovis	} Ocular Nocardiasis
Nocardia israeli	
Cohnistreptothrix foersteri	
Nocardia dassonvillei	
Monilia albicans and other species of the same genus	Ocular Moniliasis
Glenospora grapha	Ocular Glenosporosis
Sporotrichum beurmanni	Ocular Sporotrichosis

## Ocular Blastomycosis

Ocular blastomycosis is seen in the form of Palpebral Blastomycosis, which begins as papules which increase in size and give rise to pustules covered with crusts and later to a warty condition which may become red moist and granular. In other cases a subdermal nodule is formed which may ulcerate. The causal agent is *Cryptococcus dermatitis* Gilchrist and Stokes 1898. The diagnosis is made by culture of the fungus.

## Ocular Trineæ

eyelids

## Ocular Aspergillosis

eyelids

## Ocular Nocardiasis

Actinomycotic conjunctivitis was first described by Demichèri in 1899

minute grains in the lachrymal sac from which they can easily be removed

## Ocular Moniliasis

eyelids

**Ocular Glaucomatosis.**

So far, only one case in the cornea is recorded. It was found in 1910 by Morax

**Ocular Sporotrichosis.**

Sporotrichal infection of the eyelids conjunctiva lachrymal sac, and iris have been recorded but are as yet rare. The first case was found in 1905 by Danlos and Blanc the second case in 1908, the third in 1909. Four cases were recorded in 1910, and three in 1911, and several in 1912.

**DISEASES OF THE EAR.**

The diseases of the ear in the tropics require more attention than has hitherto been bestowed upon them. The majority are believed to resemble those found in the Temperate Zone. A few of the auricle are complicated

**The Auricle.**

Deformities of the lobule are common among Indian girls and

Blond children and others as occurring among the Masai and the

and in 1835, Bramley, drew in of the auricle which was tuate in one of the Nepal

the size of a pigeon's egg in four to eight weeks. It is usually

of  
ish  
fa  
ha  
me  
or

absorbed, while the skin of the ear is much thickened and shapeless. Usually both ears are affected and sometimes a succession of tumours, one after the other, may take place. The causation is unknown. It is more common in women than in men. Campbell thinks that it occurs associated with goitre, which would suggest a parasitic causation but Bramley is opposed to this view.

It is curious that there is so little literature on this subject, and that neither Scheube nor ourselves are acquainted with any further papers describing this disease.

of the ear often on both ears is reported in about or a child's head and bore their ears with thorns tained in the lobe of the ear

for some time

**Fibromata.**—We have often met with soft small fibromata attached to the lobules of the ears of the negroes on the Gold Coast which can readily be removed. Perhaps the lipomata of

form large tumours

### External Auditory Meatus

**Foreign Bodies** are commonly met with in the tropics and may consist of animate objects such as beetles flies etc or inanimate objects such as pieces of wood etc. Usually their presence can easily be determined by inspection which should always be carried out before any treatment is resorted to. The first treatment should be to attempt to wash the body out of the meatus by means of hot boracic lotion and an ear syringe unless the body be a pea bean or grain of maize because the e would become swollen with the water. Under these circumstances it is better to instil some glycerine mixed with a little rectified spirit and solution of cocaine, which causes them to contract, when a camel's hair brush dipped

in the case of impacted steel bodies the electro-magnet. If there is much swelling of the m. a. m. b.

of the ear is seen to be a ... 1911 ... Derm. ... Dermidex



These fungi cause mild inflammations of the external auditory

deafness

On inspecting the ear it will be seen that the wall of the auditory meatus is covered with a white or black macerated mucosa. The

alcohol (1 part) once or twice a day, followed by the insufflation of boracic powder, or, if there is eczema, some boracic ointment. Diamond recommends injecting a few drops of a dilute solution of sodium iodide, followed by the injection of the same amount of 12 volumes hydrogen peroxide

meatus

If the plug is soft, it can easily be removed by warm syringing, if hard, it requires to be softened by a warm solution of bicarbonate of soda (20 grains to 1 ounce of water) or glycerine of borax, and then to be removed by syringing. If deafness persists after the

pyogenic germs into a sebaceous gland or hair follicle are not rare. They usually cause much pain which is aggravated by moving the jaw or touching the auricle, and may cause toothache, salivation and vertigo.

The treatment should be to relieve pain by cocaine and the meatus should be lightly packed with gauze soaked in weak carbolic

**Otitis Externa Ossificans.**—Müller has described a diffuse inflammation of the mucosa of the external auditory meatus in the tropics which often invades the periosteum and leads to ossification, and may cause exostosis

**Deaf and Dumb**

The number of deaf and dumb people is relatively high in certain parts of the tropics—*e.g.* in Ceylon there are 9 deaf and dumb males and 7 females out of 10 000 persons of each sex. In India there are 6 males and 4 females out of 10 000 persons of each sex. There is a deaf and dumb school in Ceylon.

**Ménière's Disease.**

are useful

**EAR COMPLICATIONS OF TROPICAL DISEASES.****Leprosy**

The lobules of the ear are especially liable to be attacked in tubercular leprosy and are usually left long and thickened when the disease has abated.

**Quinine**

The prolonged or considerable use of quinine may cause simple hyperæmia of the labyrinth and may be associated with middle ear congestion as described by Kirchner. The symptoms are usually tinnitus aurium and deafness. It is bilateral and comes on gradually. The quinine should be stopped and bromide of ammonium prescribed and afterwards euguinine associated with bromides may be administered.

**Arsenic**

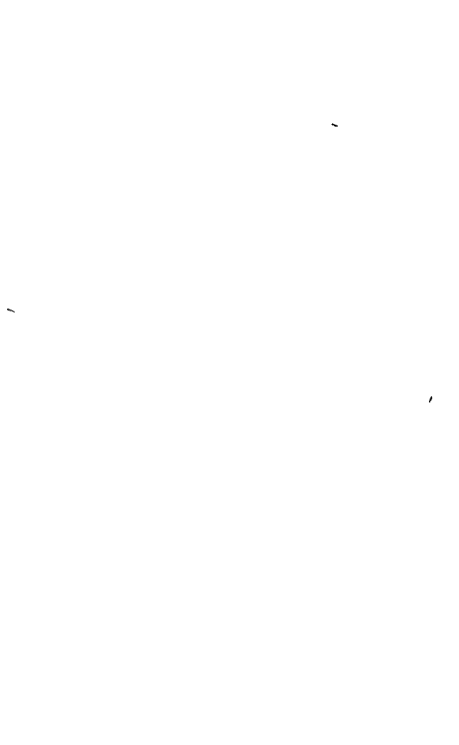
A native treatment for ear disease in the Tropics is by the application of a powder 90 to 95 per cent of arsenious acid may lead to destruction of the external auditory meatus with sometimes necrosis of the bone and even fatal hæmorrhage from the internal carotid artery.

**Malaria**

Reports of suppurative otitis media being caused by malaria cannot be accepted as proven but intermittent otalgia, intermittent attacks of deafness and labyrinthine vertigo may be of malarial origin especially if relieved by quinine.

**DISEASES OF THE NOSE.**

Disease of the nose is intimately connected with disease of the ear and to a less degree with disease of the conjunctiva. The acute catarrhal rhinitis or common cold may be induced by prolonged exposure to the sun's rays as well as to chills and is in every



## CHAPTER XXI

# PYOGENIC DERMAL INFECTIONS

Preliminary remarks—Pyogenic infections The pyoses—The pyogenic folliculites—The pyogenic dermatites—References

### PRELIMINARY REMARKS

In this chapter we begin a brief review of the more important skin diseases of the tropics. As this Manual is not a work on skin diseases

separately the dermatites due to plants, ulcers, the dermatites caused by animals, disorders of sweating and of nutrition, and miscellaneous and cosmopolitan diseases

### PYOGENIC INFECTIONS

The skin diseases which we gather together under this heading are all caused by the pyogenic cocci and may be distinguished as follows —

- A Skin between the primary lesions not inflamed —
  - I Non follicular—*The Pyoses*
  - II Follicular—*The Folliculites*
- B Skin primarily inflamed—*The Pyogenic Dermatites*

### THE PYOSES

**Definition**—A pyosis is a non follicular dermal infection characterized by the appearance on apparently healthy skin of vesicles or bullæ the contents of which rapidly become purulent. It is not associated with general symptoms except slight fever in the early stages at times and is due to the action of pyogenic micrococci.

**Remarks**—The type of this variety of dermal affections is *Pyosis mansonii* named in honour of Sir Patrick Manson GCMG.

**Varieties**—A number of pyoses are known to exist in the tropics and these may be differentiated from one another as follows —

A *Primary lesions usually small* —

- I Situate typically in axillary and crural regions with out crusty lesions—*Pyosis mansoni*
- II Situate on arms and legs with thick crusty lesions—*Pyosis tropica*
- III Situate on palms rare no crusty lesions—*Pyosis palmaris*

B *Primary lesions usually large* —

- I Typically bullæ without bright yellow crusts—*Pyosis corlettii*
- II Typically large vesicles forming circular bright yellow crusts—*Pyosis discoides*

## Pyosis Mansoni

Synonym — Pempfigus contagiosus (Manson)

Definition — *Pyosis mansoni* is a pyosis with primary lesions usually small but mixed with bullæ and affecting the axillary and



FIG 794—PYOSIS MANSONI

regions It is very common in the damp regions of

d Geographical — This dermatosis was first described  
 for the term pempfigus contagiosus It is very

common during the hot season in China the Malay Peninsula Ceylon and Southern India and probably in many other parts of the tropics. Castor has reported it from Burma. Very often epidemics occur among the crews of men of war stationed in the tropics as observed by Clayton or in offices as observed by ourselves.

*Ætiology*—Manson found a diplococcus other observers have described Leishman like bodies in Clayton's and our experience

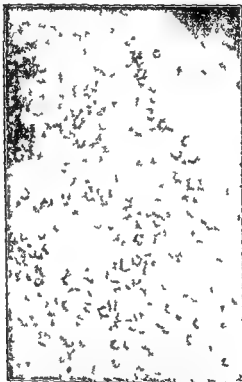


FIG 794A—PYOSIS MANSONI  
Same case as Fig 794 but more magnified

(the vesicles show the presence  
near us  
ures  
The  
hat  
erm

'pemphigus' for it is much more closely related to impetigo of which we consider it to be a variety. The eruption attacks generally the axilla the inguinal and crural regions from whence it

The eruption is made up of flattened roundish vesicles which quickly enlarge to the size of a small pea. Very large flabby pemphigoid bullæ may occur but not very frequently. The contents are at first transparent but soon become turbid. The vesicles are often surrounded by a pinkish or reddish inflammatory halo. On being pricked the vesicle collapses. The eruption does not usually affect the general health but may be very persistent and may be followed by crops of boils. As soon as the patient goes to the hills or to a cool climate it disappears spontaneously.

Diagnosis.—The eruption is generally seen in the axilla the inguinal and crural regions, the only difficulty being with

of impetigo contagiosa.

**Prognosis**—The eruption is very persistent but does not affect the general health except when complications such as boils develop.

**Treatment**—In severe cases treatment by an autogenous vaccine may be tried. Locally the affected regions should be disinfected regularly twice daily with a solution of perchloride of mercury (1 in 2000), carbolic acid (2 per cent), permanganate of potash (1 in 4000), hydrogen peroxide (10 per cent), cyllin (1 in 300), lysol or lysoform (2 to 5 per cent). After this the vesicles are pricked and the parts again washed with the disinfectant, an antiseptic powder being then thickly applied such as—

Veroform	5ʳ
Acid. borici (finely powdered)	5ʳ
Talci ven	3ʳ
Europhen	5ʳ
Talci ven	3ʳ
Dermatol	5ʳ
Talci ven	3ʳ

In some patients an ointment (europhen 2 per cent, protargol 5 per cent) answers better. The protargol ointment should never be used for the face in Europeans as it discolours the skin after a time. It may be noted that in our experience the white precipitate ointment (1 per cent) which is so efficacious in the usual impetigo

of the Temperate Zones has very little or no effect in pyosis man-  
soni. After the eruption has disappeared it is advisable to use  
some Condy's fluid or other disinfectant in the bath to prevent  
relapse and if the skin is not too tender a formalin soap may be  
regularly used. The underclothing should be regularly dusted with  
one of the above powders.

### Pyosis Tropica

**Synonym**—Pyosis Castellani, Kurunegala ulcers, Pyosis Caffra.

**Definition**—Pyosis tropica is characterized by the presence of  
numerous crusty lesions on the legs and arms caused by pyococci.

**Historical**—Under the name  
pyosis tropica Castellani de-  
scribed in 1909 a skin disease very  
common in Ceylon and Southern  
India. In Ceylon it is called by the  
natives kurunegala sore as it is  
especially common in that district.  
Pyosis tropica has been reported  
from other tropical countries. It  
has been observed in 1912 in  
Tripoli by Gabbi and Sabella in  
the Anglo-Egyptian Sudan by

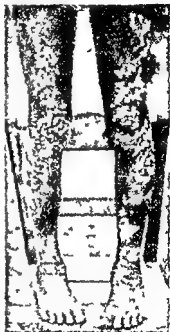


FIG. 05.—PYOSIS TROPICA

The white patches are, in reality  
thick yellow crusts.

coeci and is allied to the ordinary  
impetiginous and ecthymatous  
conditions though much more  
severe. Chalmers and O'Farrell  
grow an organism from their case  
which differing somewhat from  
the other species of *urococcus*  
was called by them *urococcus*  
*tropicus*. It was held to be causal  
because a vaccine made from it  
rapidly cured the case but had no  
effect on a case of Nile boils but  
in other cases other varieties of  
pyogenic coeci may be causal.

colour. If the thick crust is removed a shallow ulcer with an  
irregular margin and granulating fundus will be seen or a small  
flattened or hemispheric nodule the size of a pea with a pinkish



smooth surface Besides these large lesions small papulo vesicles and minute pustules are often present The larger lesions are frequently surrounded by a halo of hyperpigmentation There is severe pruritus On healing also the ulcers and nodules leave a zone of hyperpigmentation or more rarely depigmentation. In

isease might be easily mistaken for framboesia. In contrast to framboesia the disease is generally lesions as it is distinguished by the absence of burrows and absence of the *Acarus* From ecthyma to which it is closely related the condition differs by active lesions

the general

**Treatment**—If the disease is not properly treated it has a tendency to become chronic In some cases the opsonic treatment gives satisfactory results The vaccine should be prepared with staphylococci grown from the lesions It has been used by Castellani with good results in Ceylon in 1910 by Chalmers and O Farrell in 1913 and by Piper in 1918 Chalmers and O Farrell gave with success 250 millions of an autogenous vaccine to an adult and repeated it in three days Piper treated children with 4 to 5 millions of an autogenous streptococcus obtained from a case or by a mixture of streptococci and staphylococci The local treatment consists in removing the crusts by soaking them with a salicylic oil made according to the following formula —

Acid salicylic	gr xxx
Oleum cini	ʒ ss
Oleum olivæ	ad ʒiv

or by compresses soaked in hot boric lotion

All the lesions are then disinfected with a solution of perchloride of mercury (1 in 1000) carbolic acid (2 per cent) cyllin (1 in 300) or permanganate of potash (1 in 4000) A slightly antiseptic ointment is then applied such as eucrophen (2 per cent) iodoform (2 per cent) protargol (5 per cent) calomel (5 to 10 per cent) An ointment which in our experience is often efficacious is  $\beta$  naphthol gr ii v acid carbolic gr v x vaselin ad ʒi

### Pyosis Palmaris

**Definition**—Pyosis palmaris is characterized by the presence of numerous discrete conical white pustules which do not form  
by

**Climatology.**—So far it has only been recorded in Ceylon

**Ætiology.**—Pyococci are present in the lesions, but may not be the true cause

**Symptomatology.**—With little or no pruritus, discrete, conical, solitary pustules appear on the palms of the hands. These pustules do not coalesce or form crusts, and are not surrounded by a zone of hyperæmia.

ice of  
from

### Pyosis Corletti.

**Synonyms.**—Impetigo bullosa, Impetigo contagiosa bullosa of Corlett.

**Definition.**—Pyosis corletti is an acute contagious bullous pyosis beginning on any region of the body and characterized by the presence of medium sized and large bullæ arising on seemingly healthy skin, and caused by '*Styrococcus' mallis* (Dvar, 1895)

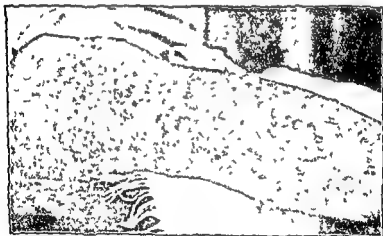


FIG 796.—PYOSIS CORLETTI

Histological. — See Corlett, J. — L. J.

smooth surface Besides these large lesions small papulo-vesicles and minute pustules are often present The larger lesions are frequently surrounded by a halo of hyperpigmentation There is severe pruritus On healing also the ulcers and nodules leave a zone of hyperpigmentation or more rarely depigmentation Invelop especially

disease might be easily mistaken for frambœsia In contrast to frambœsia the ulcers are superficial and the nodules when present are generally smaller have no moriform surface and in scrapings from the lesions the *Treponema pertenue* Castellani is absent From scabies it is distinguished by the absence of burrows and absence of the *Acarus* From ecthyma to which it is closely related the condition differs by

**Treatment**—If the disease is not properly treated it has a tendency to become chronic In some cases the opsonic treatment gives satisfactory results The vaccine should be prepared with staphylococci grown from the lesions It has been used by Castellani with good results in Ceylon in 1910 by Chalmers and O Farrell in 1913 and by Pijper in 1918 Chalmers and O Farrell gave with success 250 millions of an autogenous vaccine to an adult and repeated it in three days Pijper treated children with 4 to 5 millions of an autogenous streptococcus obtained from a case or b treatment salicylic c

Acid salicylic  
Oleum ricini  
Oleum olivæ

gr xxx  
q s  
ad ʒ v

or permanganate of potash (1 in 4000) A slightly antiseptic (1 in 300) iodiform (cent) β naph

### Pyosis Palmaris

**Definition**—Pyosis palmaris is characterized by the presence of numerous discrete conical white pustules which do not form by

**Climatology**—So far it has only been recorded in Ceylon

**Ætiology**—Pyococci are present in the lesions but may not be the true cause

**Symptomatology**—With little or no pruritus discrete conical solitary pustules appear on the palms of the hands. These pustules do not coalesce or form crusts and are not surrounded by a zone of hyperæmia.

**Diagnosis**—It is distinguished from scabies by the absence of the acarus from ringworm by the absence of a fungus and from syphilis by the uselessness of the specific treatment.

**Treatment**—Vaccines may be tried

### Pyosis Corletti

**Synonyms**—Impetigo bullosa Impetigo contagiosa bullosa of Corlett

**Definition**—  
beginning on  
presence of  
healthy skin



FIG. 796.—PYOSIS CORLETTI

**Historical**—In 1899 Corlett described a contagious bullous eruption as being endemic in Florida; later Singh in India invited attention to a similar disease. In 1912 Reguiz described an epidemic among Europeans in Cairo. In 1915 Chalmers and O'Connor gave a description of an epidemic of this disease as seen in the 1st Battalion of the Suffolk Regiment in Khartoum.

**Ætiology**—The causal organism so far found is *Aurococcus mollis* (Dix 1905). It is causal because (1) it is the only organism present and is found in the youngest vesicles (2) it was obtained,

from all the cases of the Khartoum epidemic and a similar organism was found by Corlett but not fully defined (3) a vaccine prepared

The Khartoum epidemic was traceable to a case of Nile boils

which is so quickly followed by an outbreak of bullæ that the eruption is well developed in two days

The essential feature of the eruption is a bulla arising on apparently healthy skin and measuring about 2 cm in diameter

size until it forms a bulla the walls of which are first tense and the contents watery but later they become flaccid and the contents purulent

The bulla bursts the contents escape and the lesions dry up and disappear usually without forming a scab but in the case of the larger lesions it leaves behind it a certain amount of dark discoloration of the skin indicating the affected area

If a bulla is pricked it is found to have a glazed parchment like

are rare and when present only slightly developed

The bullæ are situated most abundantly on the thighs back and chest and less abundantly on the neck arms and legs and more rarely on the face and head The axillary and scrotocrural regions

complain of a slight amount of itching which is probably due not so much to the eruption itself as to the rubbing of the clothing producing slightly raw areas where bullæ have burst When this takes place small scabs are apt to form especially if the patient scratches the area but they are entirely secondary in nature and not part of the true eruption

The differential leucocyte count based on 1000 cells is --

Polymorphonuclear leucocytes	86.7
Mononuclear leucocytes	3.8
Large lymphocytes	4.4
Small lymphocytes	3.2
Eosinophile leucocytes	9
<b>Total</b>	<b>100.0</b>

absence of crusts and of streptococci the presence of *Aurococcus mollis* and finally the ready reaction to treatment by a vaccine prepared from this organism

**Differential Diagnosis**—The differential diagnosis must be made from *impetigo contagiosa*, *dermatitis bullosa plantaris*, *pemphigus acutus* and *pyosis mansonii*

It can readily be differentiated from *impetigo contagiosa* by the absence of crusty lesions as a rule and by the fact that even when the youngest vesicle is examined by Sabouraud's methods no streptococcus can be found and only *Aurococcus mollis* (Dyar)

From *dermatitis bullosa plantaris* it may be distinguished by not attacking the soles of the feet as far as has been recorded by not extending between the toes and by the absence of streptococci and *Epidermophyton cruris* Castellani

From *pemphigus acutus* it can be recognized by the absence of the severe constitutional disturbance

From *pyosis mansonii* it can be differentiated by the fact that it does not begin in the axillæ or scroto-crural regions and that it but rarely and then lightly attacks those parts which are the

large junctions where are common in *Manson's pyosis* the vesicles are often surrounded by a pinkish or reddish inflammatory halo which is absent in the present eruption

**Complication**—Eruptions of boils may occur

**Sequela**—When cases are not treated by vaccine therapy there rapidly

vaccine which is to be administered in 200 and 450 million doses with intervals of two to three days between each dose

In order to expedite the cure local treatment is also useful and this consists in pricking each blister and catching the exuding fluid on swabs dipped in 1 in 1000 lotio hydrargyri perchloridi

The term eczema should only be used when the practitioner is unable to assign its proper cause to a dermatitis. The term is there-

subclass which is brought about by the pyococci

We however propose to further restrict our remarks to merely the streptococcal dermatitis and will consider them under two headings—viz —

- Primary Streptococcal Dermatitis
- Secondary Streptococcal Dermatitis

### THE PRIMARY STREPTOCOCCAL DERMATITES

**Definition** — A primary streptococcal dermatitis is an inflammation predominantly of the surface of the skin localized or generalized and caused by streptococci

**Remarks** — Streptococci appear to have been first observed in cutaneous lesions by Crocker in 1881 and to have been later identified by Brockhart as *Streptococcus erysipelatosus* Fehleisen 1883. Later they were carefully described by Whitfield Colcott Fox and many other observers including ourselves. Colcott Fox classified streptococcal skin lesions with those which occur in the course of grave systemic affections, those which are lesions of the hypoderm, those which are primary and those which are secondary cutaneous lesions.

With regard to the primary streptococcal dermatitis these are sufficiently numerous but for our present purpose we will restrict our attention to those found in the tropics which are *dermatitis veldis*, *dermatitis pratensis* and *dermatitis cupuliformis* which may be distinguished from one another as follows —

#### A. Nodules not produced —

I Begins as a large blister or bulla and forms a superficial sore with ragged edges and a fresh clean floor in recent or a parchment like floor in old cases—  
*Dermatitis veldis*

II Begins as a small irritable papule or papulo-vesicle which ulcerates glazes over spreads and finally

#### *pratensis*

#### B. Nodules produced—*Dermatitis cupuliformis*

#### *Dermatitis Veldis*

**Synonyms** — Veld sore Barcoo rot Gift zeer Brand zeer

**Definition** — A primary streptococcal dermatitis characterized by the formation of a bulla followed by a superficial sore which

becomes very chronic, but does not lead to papillary acanthotic formations

'brand zeer' or burn sore

from the one in Australia and from the other in South Africa recognizing them as the same. In 1913 Black gave us his personal experiences of Barcoo rot. In 1917 Martin met with a similar

as the causal organism and called it *Micrococcus vesicans*, but it is more probable that the causal agent is the streptococcus which may be known provisionally as *Streptococcus vesicans*, and which may be only a synonym for some more fully worked out species such as *S. versatilis* Broadhurst, 1915. The disease can be reproduced in man by inoculating the fluid of the blisters on to a raw place in the skin.

**Pathological Histology.**—The fresh unopened blister has been

somewhat festooned margins

The floor of the ulcer remains dry like parchment. The ulcer may be painful, but, especially at the beginning, there is frequently



more itching than pain. The proximal lymphatic glands may be enlarged. Several such sores may be present at the same time. The hands, forearms, feet and legs are mostly affected. Harman has seen as many as twenty such sores on one man. The duration of the affection varies between one and three months, but occasionally may last as long as six months.

**Treatment**—The correct treatment is by vaccines, either autogenous or from a locally prepared stock. The local treatment consists in keeping the sores dressed with antiseptic lotions, such as perchloride of mercury (1 in 4000) or in washing the sores with a disinfecting lotion, and then applying an antiseptic powder, paste or ointment, such as eucrophen (1 to 3 per cent), iodoform (1 to 3 per cent), protargol (5 to 10 per cent) or calomel (5 to 10 per cent).

### Barcoo Rot

For the reasons given above we consider Barcoo rot to be a condition identical with *dermatitis veldis*. It must however be admitted that the descriptions given by some authors may include other conditions. For example Black described it as forming a crust which becomes larger, thicker and harder until horny in consistence and difficult to remove. There is very little itching or pain.

Pain states that the term *Barcoo Rot* is applied to him the original Barcoo conditions of living scurvy, the name to any superficial condition now covered by the term *dermatitis veldis*, a seasonal staphylococcal infection occurring generally in the autumn, and a deep-seated trichophytosis of the hands.

### Dermatitis Pratensis

torial Africa and by Chalmers and Archibald in the Anglo Egyptian Sudan. We give it the name *dermatitis pratensis* to bring it into line with the other *dermatites*.

**Ætiology**—The causative organism is a streptococcus which can be found in the polymorphonuclear leucocytes in the lesions and cases in the Anglo-Egyptian Sudan. It is a *Streptococcus versatilis* from equine faeces from

covered by masses of scales, both on its free surface and on the surface looking towards another process.

This epidermis shows the defect of cornification called 'parakeratosis' by Auspitz, while with regard to the rete there is a certain degree of acanthosis.

The corium is full of dilated vessels, and is also slightly cedematous and fairly cellular.



FIG. 799—DERMATITIS PRATENSIS

Turning now to the non-papillomatous regions, there is a well defined epidermis with parakeratosis and acanthosis, while the cellular infiltration of the corium is denser. It is somewhat cedematous.

exudate.

leucocytes in certain regions and more especially superficially marked

In the deeper part of the section isolated pieces of the ordinary connective tissue of the corium can be seen while finally in the depth of the section well defined connective tissue is seen contained here and there scattered collections of cells of the same nature already described for other portions of the tissue

over and extends at its margins. When fully developed it is surrounded by a raised margin behind which small papillæ may be noted which in older cases give rise to very distinct papillomatous outgrowths. The surface of the sore is composed of deep fissures and a few ulcerated areas which exude a serous fluid which is apt to form crusts. These ulcerated areas and fissures are separated by other areas coated by a thin epidermal covering which gives rise to a false peeling appearance. The whole condition spreads slowly from the margin.

**Diagnosis**—This is sufficiently effected by the table given on p. 2030.

**Prognosis**—The prognosis is good provided that the patient is otherwise healthy.

**Treatment**—The best treatment is without doubt an autogenous vaccine but a polyvalent local (made from local strain) vaccine acts quite well.

We generally give 10 millions to commence with then 50 and if necessary 100 millions.

The affected part in chronic cases may be painted with iodine and a dry dressing applied.

### Dermatitis Cupuliformis

**Synonym**—Tropical ecthyma (Castellani)

**Definition**—Dermatitis cupuliformis is characterized by commencing as dusky red macules which are follicular or perifollicular which either disappear or slowly become cupuliform nodules which

in 1914  
he me

and the

as superficial dusky red  
 ung macules on the feet and

edges

These ulcers are somewhat painful and very slow to heal and when this does take place it produces patches of hyperpigmentation. The course of the disease is very long lasting at times more than a year.

Diagnosis—The characteristic features of the eruption are the presence of raised hard rather large cupoliform nodules some of which show a central ulcer with undermined edges.

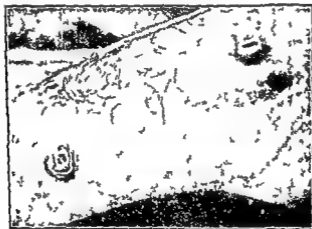


FIG. 800.—DERMATITIS CUPOLIFORMIS OF THE FOOT

The differential diagnosis has to be made from *Oriental sore* by the absence of Leishman Donovan bodies.

From *ecthyma* it may be distinguished by the absence or rarity of the pustular lesions with a brownish crust. In *ecthyma* the initial lesions are always pustular there being generally discrete flat pustules when these rupture a brownish crust is formed beneath

ly pustules

is difficult

to cure, unless treated by an autogenous vaccine

**Treatment.**—An autogenous vaccine is the correct treatment and generally produces a cure in two to three weeks. Local treatment by antiseptic lotions is also recommended.

### THE SECONDARY STREPTOCOCCAL DERMATITES

be differentiated as follows —

- A Situate primarily between the toes Bullæ absent—*Dermatitis interdigitalis*  
 B Situate primarily on the soles Bullæ present—*Dermatitis bullosa plantaris*

#### **Dermatitis Interdigitalis.**

**Synonyms**—*Dermatitis rimosa* of the toes Mango toe (Ceylon) Frieira (Brazil)

**Definition**—*Dermatitis interdigitalis* is a streptococcal dermatitis secondary to an infection of the parts between the toes caused by *Epidermophyton cruris* Castellani

**Historical**—This affection which is popularly known in Ceylon as 'Mango toe' was first brought into prominence by Sabouraud's observation that the primary cause was an infection by *Epidermophyton cruris* Castellani. In 1910 Castellani found that very often there was a secondary streptococcal infection.

A somewhat similar affection was described long ago by Martin Costa in Brazil who stated that the condition was very common among natives, who called it 'frieira'. He believed it to be caused by the heavy perspiration and accumulation of dust and dirt between the toes.

**Geographical Distribution**—It is extremely common in Southern India, Burma, Ceylon and many other tropical countries, being the cause of great discomfort to European residents especially during the hot season.

**Ætiology**—The condition seems to be a pyogenic infection starting generally on slight lesions produced by a localization of *Epidermophyton cruris* Castellani to the toes. This localization of the fungus was first observed by Sabouraud. The fungus *per se* very often only

of great itching  
any papular or

vesicular lesion. On scratching to relieve this itching portions

## DERMATITIS BULLOSA PLANTARIS

toes become removed, and small, superficial red, irritable  
become severer, and deep, extremely pain-  
ful in most all the cases. This  
the patient

2

going to periods and then reappears  
some pruritus may occasionally be  
toes may easily crack or be slightly scaly  
Treatment.—This consists in keeping the patient at rest for a few  
days dressing the affected parts continuously with diluted carbolic  
lotion (½ per cent) or resorcin lotion (½ to 1 per cent), and later  
applying a zinc oxide paste hazeline cream, or a bismuth borac  
ointment (bismuth subnitrat, gr xxx, acidi borici gr xv,  
vaseline, lanoline aa ʒiv) The stockings should be white, and  
should be changed at least twice daily and should be boiled before  
use. If a fungus is found in the lesions an antimycotic treatment  
should be carried out when the acute stage is over or during the  
quiescent periods by means of silver nitrate (3 per cent) or a  
strong solution of potassium permanganate (gr xxv to ʒi),  
painted on once or twice daily, or tincture of iodine may be used  
if the lesions are dry and there are no excoriations.

### Dermatitis Bullosa Plantaris.

Synonym.—Foot tetter (Cantlie)

Definition.—Dermatitis bullosa plantaris is a streptococcal  
dermatitis often secondary to an infection of the soles of the feet,  
caused by *Epidermophyton cruris* Castellani

Historical and Geographical Distribution.—This affection was  
first described by Cantlie in China but cases are met with in  
tropical countries

Etiology.—The disease is probably a streptococcus infection  
one of us having isolated a very virulent strain of the germ from  
the blebs of a number of cases In several of our cases this strep-  
coccus infection developed on some superficial lesions due to  
localization of *Epidermophyton cruris* Castellani to the soles

Symptomatology.—It commences with blebs on the sole of  
foot The blebs ultimately break and by and-by bare scaly  
of skin form and extend all over the sole and between the  
There is intense itching Occasionally the condition spreads to  
parts of the body It usually dies away in the cold season  
recommences in the hot season According to Cantlie persons  
who have returned to Europe see their affection reappearing  
fully every summer for ten or twenty years after leaving the  
Prognosis.—The condition is of difficult cure and relapses  
Treatment.—In the acute stage the patient must stay in  
complete rest, and antiseptic dressings such as mercury pre-  
parations (ʒi in 4000) or carbolic acid (½ per cent) applied con-  
tinuously with a lead lotion (liq plumbi. ʒii, aq destil. ad ʒv)

later some antiseptic powder (dermatol or zinc oxide) may be used. In our cases ointments were always badly borne. In

If the eruption develops on lesions due to fungi, which may be carried out with a silver nitrate or tincture of

iodine if the lesions are dry

Cantlie recommends the application of pastes and plasters as palliatives. Manson advises the use of a daily foot bath of a 2 per cent solution of carbolic acid for half an hour.

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## CHAPTER XCII

### TROPICAL DERMATOMYCOSES

TROPICAL dermatomycoses—that is to say tropical skin diseases caused by fungi higher than bacteria—may be classified as follows—

#### TROPICAL DERMATOMYCOSES

- Ep cruris* Castellani 1905 common variety of *Tinea cruris* (d'hoobie itch)
- Ep pcrneta* Castellani 1907 variety of *Tinea cruris*
- Ep rubrum* Castellani 1909 variety of *Tinea cruris*
- T nodiformans* Castellani 1911 variety of *Tinea cruris*
- T macfadyens* Castellani 1905 variety of *Tinea alba*
- T albiscicans* Næuwenhuis 1907 *Tinea albigena*

I D . . . of the . . . variety of *Tinea capitis* nigro

- Castellani 1911
- T curvis* Chalmers and Marshall 1914
- T discoides* Sabouraud 1909
- T violaceum* Bodin 1902 var *haar*
- loumense* Chalmers and Macdonald 1915

Varieties of *Tinea capitis* variety

- II Due to fungi of the genus *Endodermophyton* Castellani 1909
- En tropicale* Castellani 1914 *Tinea imbricata*
  - En indicum* Castellani 1911 *Tinea imbricata*
  - En castellani* Perry 1907 *Tinea intersecta* 2040

# TROPICAL DERMATOMYCOSES

- II Due to fungi of the genus *Malassezia* Baillon 1889 } *M. tropica* Castellani 1905 *Tinea flava*
- IV Due to fungi of the genus *Cladosporium* Link 1809 } *C. mansoni* Castellani 1905 *Tinea nigra*  
*C. madagascariensis* Verdun 1913 peculiar nodular affection
- V Due to fungi of the genera *Saccharomyces* Meyen 1838 *Cryptococcus* Kutzinger and Gilchrist 1897 *Mortierella* Persoon 1797 } Several species some of which incompletely investigated } Varieties of blastomycosis
- VI Due to fungi of the genus *Nocardia sensu lato* Tonia and Trevisan 1889 and Collins *streptothrix* Pinoy 1911 } *N. minutissima* Burchardt 1859 erythrasma  
*N. carougeana* Brumpt 1910 juxta articular nodules  
*N. rivieri* Verdun 1912 nodular affection  
*N. tenuis* Castellani 1912 trichomycosis axillarum  
*N. bergesii* Pinoy and Ravaut 1909 nodular
- VII Due to fungi of the genera *Sporotrichum* Link 1809 *Hemispora* Vuillemin 1906 *Enantiothamnia* Pinoy 1911 *Scopulariopsis* Basnier 1907 } *S. schenckii* 1909  
*S. asteroides* Splendore 1911 } Various types of dermatomycosis  
*S. indicum* Castellani 1908  
*Hemispora stellata* Vuillemin 1906  
*" thamnia brevifolia* Pinoy
- VIII Due to fungi of the genera *Aspergillus* Michels 1725 *Sterigmatalocystis* Cramer 1869 *Madurella* Brumpt 1905 *Indiella* Brumpt 1906 *I. reymiersii* Brumpt 1906 *I. somaliensis* Brumpt 1906 *Nocardia tonia* and Trevisan 1889 Collins *streptothrix* Pinoy 1911 } *Cladospium* Verdun 1913  
*Acladium castellanii* Pinoy 1910  
*Aspergillus bouffardi* Brumpt 1906  
*Sterigmatalocystis nodulans* Eidam 1883  
*Madurella mycetozoa* Laveran 1902  
*M. bovis* Brumpt 1910  
*M. toxeus* Nicolle and Pinoy 1906  
*Indiella mansoni* Brumpt 1906  
*I. reymiersii* Brumpt 1906  
*I. somaliensis* Brumpt 1906  
*Nocardia madura* Vincent 1894  
*N. asteroides* Gppinger 1891  
*N. pelleterii* Laveran 1906  
*N. bovis* Harz 1877  
*C. israeli* Kruse 1896  
*Sporotrichum brumptii* Matruchot and Ramon 1905  
*Monosporium apiospermum* Saccardo 1911  
*Glenospora tharomycensis* Chalmers and Archibald 1916  
*G. zimmermanni* Chalmers and Archibald 1917
- IX Due to fungi of the genera *Aspergillus* Michels 1727 Link 1809 } *A. barbae* Castellani 1907 hairy parts  
*A. barbae* Castellani 1907 Penicillium hairy parts

- |  |   |   |
|--|---|---|
| X. Due to fungi of the genera                                    | $\left\{ \begin{array}{l} \textit{Aspergillus} \textit{ Micheli} \textit{ 1725} \\ \textit{Penicillium} \textit{ Link} \textit{ 1809} \\ \textit{Monilia} \textit{ Persoon} \textit{ 1791} \\ \textit{Montoyella} \textit{ Castellani} \textit{ 1907} \end{array} \right\}$ | Pinta                                       |
| XI Due to fungi of the genus <i>Trichosporum</i> Behrend 1890    | $\left\{ \begin{array}{l} \textit{T giganteum} \textit{ Behrend} \textit{ piedra} \\ \text{Species as yet not well determined} \end{array} \right\}$  | Tropical varieties of nodular trichomycosis |
| XII Due to fungi of the genus <i>Pityrosporum</i> Sabouraud 1903 | $\left\{ \begin{array}{l} \textit{Pityrosporum canaliculatum} \textit{ Castellani} \textit{ 1907} \textit{ variety} \\ \text{of tropical seborrhoea} \end{array} \right\}$  |   |

From the above table it will be seen that tropical dermatomycoses *sensu stricto*—viz occurring only in the tropics—are comparatively few. Most of them are endemic also in temperate zones though occurring there rarely or at any rate less frequently than in the tropics. We may mention as examples tinea cruris and Madura foot. The same remark however applies to every other branch of tropical medicine.

The frequency of dermatomycoses in the tropics is probably due to the hot damp climate being very favourable to the growth of vegetal parasites.

### TINEA CRURIS (DHOBBIE ITCH).

**Synonyms**—Tinea tropicalis Tinea inguinalis Tinea axillaris, Eczema marginatum

**Definition**—The term *tinea cruris* indicates a group of epidermophytoses and trichophytoses which are clinically characterized by their tendency to develop on the scroto-crural and inguinal regions.

**Historical and Geographical**—Tinea cruris is extremely common all over the tropics. It is met with also in subtropical regions and

by the yellowish cultures. Castellani used the name *tinea cruris*. Pernet found and described a fungus for which Castellani

and Pinoy having isolated in Ceylon with those found in France have found them identical.

Castellani's further researches have shown that other fungi besides *Ep. cruris* may give rise to tinea cruris each species giving rise to a slightly different clinical variety of the eruption.

**Ætiology.**—According to Castellani's researches at least three different species of *Epidermophyton* gave rise to the eruptive form. Castellani, *Epidermophyton formans* Castellani—an undescribed species.

The description of these fungi is given in Chapter LXXVIII (see p. 1024). Attempts at experimental reproduction of the disease made by Sabouraud and one of us in human beings and monkeys have failed.

women the regions under the breasts. It is in this stricter meaning that the term is used by medical men practising in the tropics.

The clinical features of the affection correspond to Hebra's 'eczema marginatum'. In a well marked case the perineum

The disease if not properly treated is extremely chronic, the condition improves during the cold season but gets worse again during the hot months. Patients who suffer badly from dhobie itch may get almost well in a few days without any treatment on going to the hills; on returning to the plains the pruritus and all

trunk, legs, etc. In such situations it may develop in rings or  
ma  
be  
son  
and  
inguinal regions.

**Clinical Varieties.**—The above description mostly applies to the  
1 is the com  
es due to *Ep*  
ry beginning  
so raised as  
of numerous  
minute bloody  
be present in  
the shape of large complete or incomplete gyration enclosing normal

skin, or solid patches may be seen. The variety due to *Ep. rubrum* has great tendency to spread to other regions of the body.

In the cases caused by *Trichophyton nodiformans* the eruption

typical lesion varie due to the same organism on the foot skin.



FIG 801.—DHOBIE ITCH OF THE CRURO INGUINAL REGIONS. *TINEA CRURIS*.  
From the same case (due to *Epidermophyton cruris* Castellani) as the  
coloured plate

**Regions of the Body affected.**—The eruption, as already stated,  
regions  
ne fungi  
part of  
20 703

first appear on  
and armpits,  
'tinea cruris

appropriate, and the term 'tinea tropicalis,' or the native term 'dhoobie  
itch,' might be used as general terms to cover all the localizations.

A localization of great importance noted by Sabouraud, and later





# COMMUNICABILITY

Whitfield is when the fungi invade the skin between the toes  
 (Tinea interdigitalis) In this situation the fungus—it is generally  
 practically no objective symptom except perhaps a little scaliness  
 but generally induces erythema pruritus especially in the hot  
 weather. The latter is often the starting point  
 for the infection. The fungus is most distressing der-  
 matitis known to the public as the Fungus toe and  
 itchy foot.



...the will be (n  
 ...are not  
 ...the f  
 ...two of  
 ...me  
 ...n  
 ...ff  
 ...ng them  
 ...again affect th  
 ...the clothes



or in small pools of water more or less stagnant. It is certain that clothes belonging to infected persons are washed together with other clothes. Dhobie itch is very contagious; true epidemics occur in schools and among soldiers in barracks.

**Prognosis**—If the affection is not energetically treated it has a tendency to become very chronic and last for years. Occasionally the eruption spreads to the whole body forming rings or solid patches; at other times a distressing dermatitis develops on old dhobie itch patches due to scratching. Tinea cruris may disappear during the cold season or when the patient goes to the hills only to reappear as soon as the hot season commences. During the period of quiescence the skin of the affected regions often shows a brownish discoloration, furfuraceous somewhat



FIG 803—DHOBIE ITCH OF THE AXILLARY REGIONS  
TINEA AXILLARIS

Case due to *Epidermophyton cruris* Castellani

edge being quite typical. In old cases especially when secondary lesions due to scratching are present the diagnosis may be very difficult, the affection being often mistaken for eczema.

In doubtful cases the microscopical examination will be of great help. It must however be noted that in old cases the fungus may be extremely scarce, the mycelium being practically absent and only a few spores being found; it is well to take the scrapings for microscopical examination from the edge of the eruption. The differential diagnosis must be made from ery

limited by a raised red edge the fungus *Trichophyton* ...

contour and the margin is not sensibly elevated, no epidermal ...

nt  
ed

or *Trichophyton* like fungi are found. Saccharomycetic intertrigo is rare, there is no elevated margin and the fungus is found to be a *Saccharomyces* (*S. sambonii*)

*Primary eczema* of the scrotum and of the skin of the thighs in

it from tinea cruris. As already stated however an eczematous like dermatitis due to scratching often develops after a time on old chronic itch lesions.

*Diagnosis of the Toes Localization*—The complaining of severe itching between the toes even if there are no objective symptoms whatever should in the tropics always arouse the suspicion of a possible local fungus infection especially if the patient suffers at the time or has been suffering from tinea cruris and scrapings

Treatment —

Mild Cases —

Resorcin ʒi

Tincture of iod

ice daily

āā ʒiv

a certain

amount of smarting and must be applied with care and in only very recent cases with no eczematoid lesions. In some cases we use Vlemineckx solution or lotio calcii sulphurati (slaked lime ʒ sublimed sulphur ʒ distilled water ʒ5 boil together evaporate and filter to produce 20 of solution) pure or diluted. A sodium hypsulphite solution (sodium hyposulphite ʒii aq ʒi) may also be used.

Severe Cases —

Chrysarol is obtained from araroba which is known by the name of goa powder all over the East. The crude goa powder partly dissolved in vinegar is often used but frequently induces very severe inflammatory symptoms.

In obstinate cases we use local applications of turpentine-oil in the morning and at night.

*Cases complicated with Eczematous Dermatitis and Fissures*—In such cases in our experience it is better to use at first a soothing treatment by lead lotion or a solution of resorcin ( $\frac{1}{2}$  to 1 per cent) or glycerin boracic in rose water with the object of first healing the eczematous lesions. Later to the rhagades which so often develop in the inguinal regions we apply a solution of nitrate of silver (arg nitr gr v xv sp æth nitr ʒi). We touch with this

destroys the fungus. At night we apply a mild ichthyol ointment (2 per cent) or hazeline cream all over the eruption as soon as the parts have become less moist we begin the chrysarobin treatment. A precaution which must always be observed during and after the treatment to prevent reinfection is to dust all the undergarments with antiseptic powder—for instance Manson's powder (ac bor zinc ox amyli aa p æq) salicylic powder (ac sal cyl gr x talci venet ʒi) menthol powder (mentl ol gr v alcohol q s talci venet ʒi) dermatol powder (dermatol gr xv talci ʒi). It is advisable to wear small bathing pants which can be washed in the house.

*Treatment of Generalized Dhobie Itch*—When the eruption is diffused all over the body the simplest treatment is as a rule the application of tr iod or lin iod treating only one portion at a time. A ch

*Treatment of*  
cent periods

tincture of iodine may be used or an alcoholic solution of salicylic acid (2 per cent). When there are acute symptoms of dermatitis the treatment must be at first a soothing one as described on p 2037.

### TINEA ALBA

Remarks—Tinea alba is in reality

ditions which give a white powdery appearance to the skin of the natives.

**Historical**—This condition was first described by Castellani in 1905 in



FIG 804.—FUNGUS FOUND IN A VARIETY OF TINEA ALBA  
*Tr. macfadyeni* CASTELLANI

Cc

1905 (*Trichophyton macfadyeni*) the description of which is given on pp 1009 and 1016

**Symptomatology**—The arms legs chest and occasionally the whole body present a diffuse eruption of white powdery appearance this being due to the very numerous small white pityriatic squamæ present. The margins of the eruption when the causative fungus is *Ep rubrum* may be distinctly raised and dotted with minute close set papules.

**Course and Prognosis**—The course is very chronic. Apparently the fungi have a disturbing action on the production of pigment in the skin and after some years white leucodermic patches may develop in which no fungus is found.

**Diagnosis**—This is based on the diffuse eruption with the abundant fine pityriatic desquamation and with well marked limits—and the microscopical



FIG 805—TINEA ALBA DUE TO *Ep of r*  
*mophyton rubrum* CASTELLANI



FIG 806—TINEA ALBA

examination. It is quite easily distinguished from *tinea imbricata* by the squamæ being pityriatic very small easily detached. In leucoderma the surface is smooth no squamæ being present and no fungus found. As already stated however if the eruption is left untreated for a long time leucodermic patches may develop.

**Treatment**—The best treatment is by a chrysarol ointment (2 to 5 per cent) applied with the precautions already mentioned. Tincture of iodine and liniment of iodine may also be used.

## TINEA ALBIGENA.

This trichophytosis was first described by Nieuwenhuis in Java. Nieuwenhuis' researches have been confirmed and enlarged by Jeanselme in Indo China and in Siam in the latter country the

very slow the colonies are whitish and show a powdery surface



FIG 807—TINEA ALBIGENA

**Symptomatology**—The eruption generally affects the palms of the hands and the soles of the feet but may extend to the forearms and legs and may affect the nails. It begins with the appearance of small pruriginous spots on the palms and soles the epidermis becomes raised and bullæ develop containing at first clear serum the bullæ break and the skin remains dry and peels

natural folds. Several horny semidetached discs can often be seen at the dilated orifices of the sweat glands. The affection is very chronic, it may begin in youth or in adult life. After some time a process of pigmentation of the skin sets in, white patches, leucoderma like, developing and extending often to the legs and arms.

**Treatment**—Tincture of iodine and chrysarobin ointment (1 to 5 per cent) answer fairly well, but the pigmented patches are not cured.

### TINEA SABOURAUDI TROPICALIS.

This trichophytosis was first described by Sabouraud in patients returning from Indo-China, Japan and Tonkin. We have seen a few cases in Ceylon.

**Ætiology**.—The disease is caused by the fungus *Trichophyton blanchardi* Castellani 1905 (synonym *T. sabouraudi* Castellani 1905). The term *T. sabouraudi* cannot be applied to this *Trichophyton* as this name has already been used for another *Trichophyton*—*T. sabouraudi* R. Blanchard 1905. This fungus cannot be grown on Sabouraud's media or any other media we have tried. Microscopically the mycelial tubes do not show a double contour, and are not very straight, they are often banana shaped. The segments of the mycelium are all separated, the mycelial spores are roundish and are shed without forming a filament by their union. They are of various sizes.

**Symptomatology**.—The eruption generally commences on the uncovered parts of the body, generally on the legs, the patients often state that they think the disease is due to prolonged immersion in stagnant water. The affection begins with erythematous patches, the surfaces of which are covered with minute pityriatic squamæ. After reaching the diameter of about 1 or 2½ inches the patches become circinate. The circination however is incomplete, it is only segmentary. In dependent positions large polycyclic patches may be seen, but only one half or one third of the circles are clearly seen, the rest being badly defined. The base of the patches at this stage is of a very dark bistre brown colour. The border shows polymorphic lesions, fine pityriatic squamæ, minute vesicles and papules. The pruritus is very marked and excoriations due to scratching are constantly present. In chronic cases a thickening of the skin with lichenification takes place, specially at the circinate border.

**Treatment**.—The disease is difficult of cure in the tropics, though it may disappear spontaneously on the patient proceeding to Europe. Chrysarobin ointment (1 to 4 per cent) is the best treatment.

## TINEA ALBIGENA.

This trichophytosis was first described by Nieuwenhuis in Java. Nieuwenhuis' researches have been confirmed and enlarged by Jeanselme in Indo China and in Siam, in the latter country the disease is known as *kh*. The disease is fairly common in the Malay

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 1907

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FIG. 807.—TINEA ALBIGENA

**Symptomatology.**—The eruption generally affects the palms of the hands and the soles of the feet but may extend to the fore-arms and legs and may affect the nails. It begins with the appearance of small pruriginous spots on the palms and soles, the epidermis becomes raised and bullæ develop containing at first clear serum, the bullæ break and the skin remains dry and peels

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natural folds Several horny semidetached discs can often be seen at the dilated orifices of the sweat glands The affection is fe After some white patches to the legs and

arms

Treatment.—Tincture of iodine and chrysarobin ointment (1 to 5 per cent) answer fairly well but the pigmented patches are not cured

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They are of various sizes.

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Europe Chrysarobin ointment (1 to 4 per cent) is the best treatment



## TINEA NIGRO-CIRCINATA

This trichophytosis has been observed by one of us among Sinhalese natives

**Ætiology**—The eruption is due to a *Trichophyton*—*T. ceylonense* Castellani 1908. The spores are very few in number, roundish rather large ( $4 \mu$ ) and showing a double contour. The mycelial tubes are about  $3\frac{1}{2} \mu$  in breadth, generally straight. The fungus does not grow on any of the ordinary or Sabouraud's media.

**Symptomatology**—The eruption is found most frequently on the neck and scrotum and consists of a few rings with thick, elevated margins, the encircled skin being black—much darker than the healthy skin—but not thickened nor presenting papules, vesicles or pustules. The edge is thick, elevated, of a dark colour with the upper portion pinkish or occasionally covered by a dark crust. The eruption often heals spontaneously, leaving dark roundish patches at the previous seat of the lesions.

**Prognosis**—The eruption may disappear spontaneously and the treatment is easy.

**Diagnosis**—The only dermatomycosis to which it has a slight resemblance is *Tinea sabouraudi*. The latter however is very chronic and invades large portions of the body; the rings are segmentary, not complete, and the edge is not so thick and elevated.

The condition may also be distinguished from a circinate frambœside or ringworm yaw, in which no fungus is found while *Treponema pertenue* Castellani is present.

**Treatment**—Tincture of iodine freely applied answers well.

## TINEA CAPITIS TROPICALIS

**Definition**—*Tinea capitis tropicalis* is ringworm of the head as seen in the tropics.

**History**—Celsus in the second chapter of the sixth book of his *De Medicina* gives an account of ringworm of the head under the name *porrigo*. His words are: *Porrigo autem est ubi inter pilos quædam quasi squamulæ surgunt, æque a cute resolvuntur et*

*defricans quicquid erat crustam scabiem tineam nec purulentam fastidians*. He uses the term *porrigo* in the sense of *genorum*.

In the tenth century Alî ben Abbas (often written Haly) who lived in Persia described the complaint under the terms *salafati* and *alvathim*.

The Anglo Saxons applied the word *teter* to any kind of skin



TINFA NI RO CJKLNATA



TINEA CAPITIS TROPICALIS

Langham's 'Garden of Health' 1633), but in or before the  
seventeenth century the word ringworm (vide Levins or Levins  
lanipulus Vocabulorum London 1570) had appeared for the  
disease tinea circinata. In 1695 Willis in his 'London Practice of  
hysick' devoted a chapter to the subject of the running scab,  
etter or ringworm

With a history such as this it is not astonishing that the early  
English writers on tropical medicine refer to the same disease as  
seen in tropical countries

Thus in 1746 in his work on the diseases of Barbados Hillary  
says that it was noticed by the first voyagers to the West Indies  
and that it probably is the same disease as that called by the natives  
cowrap. He gives a good clinical description of tinea circinata as  
he saw it in Barbados and he is supported by Wright ('Essays on  
the Malignant Fever of the West Indies') who stated that it was  
common in Jamaica

Winterbottom in 1803 under the term 'herpes' describes the  
disease in Sierra Leone calling it serpigo ringworm or tetter and  
distinguishing it from kra kra

So far it would appear as though only the body ringworm or tinea  
circinata was meant by the terms 'tetter' and 'ringworm' but in  
1817 Bateman, the pupil of Willan who completed his master's great  
work on skin diseases published an atlas on the same subject in  
which Plate XXXIX induces Sabouraud to believe that he recognized  
identity of the two conditions. In 1824 Plumbe showed that  
cultivation of ringworm of the scalp would cause ringworm of the  
body, and vice versa

These publications appear to have stirred the practitioners of the  
ropies to study the disease as it was described in India by Young,  
n 1826 and in the Malay Archipelago by Lesson in 1829

In 1832 Alibert published the first edition of his celebrated  
'Monographie des Dermatozes' which stimulated the continental  
medical mind of the day, as is reflected by Smith's description of  
the disease in Peru in 1840 and Pruner's in FEG pt, in 1847

In 1839 Schönlein discovered the fungus causing favus  
In 1842 Gruby, who had already repeated Schönlein's observa-  
tions on the parasite of favus discovered a new cryptogam in the  
barbe w/

In 1841  
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of Malmsten gave the name of Trichophyton to the p  
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These researches naturally aroused much interest and in 1855 Heymann showed that the disease existed in the East Indies but it is noticeable that he makes no mention of observing a parasite in the affection

In 1874 Blanc described the occurrence of the disease in Abyssinia where Merab states that it is very common and where it is treated by the juice from the fruits and leaves of *Bryonia dioica* and by tobacco powder as well as by sulphur ointment

In 1872 the Army Sanitation Commission induced the British Government to instruct Tilbury Fox and Farquhar to obtain a

Kimberley and Sir Alexander Armstrong this inquiry was extended to China Japan Egypt Algeria the West Indies and Honolulu and the finished report was published in 1876

The net result of this inquiry as far as ringworm was concerned was unfortunate as it led to the dogmatic assertion that the ringworm of the body in the tropics was the same as that of temperate climes

In 1873 van Leent drew attention to the large number of cases of ringworm of the head in Chinese in the island of Banka in Malaya

In 1878 Corre gave a description of the disease and its parasite as seen in Nossi Be

From 1890 the possibility of plurality in the species of *Trichophyton* was raised but it was not until Sabouraud in 1892 began those brilliant researches which he has carried on to the present day that this was definitely established In this year he showed that

were speedily confirmed by many observers among whom may be mentioned Adamson Colcott Fox Malcolm Morris White and Mibell

In 1900 Matruchot and Dasseville showed that the *Trichophyton*s were closely allied to the *Gymnoascaceæ* and that *Ctenomyces serrata* Eidam 1880 when injected into animals produced a *Trichophyton* like mycelium and eruption

The further history may perhaps be better discussed according to the countries

papers on ringworm in which the parasitic fungi were studied according to Sabouraud's classical methods. It was at this meeting that Rabello announced that he had found *T. violaceum*, *M. audouinii*, and *M. lanosum*.

In 1909 Lindenberg reported the presence of *T. sabouraudi*. Horta announced that *M. felinum* has been found in a considerable number of cases at Sao Paulo and also isolated *T. album* Sabouraud, 1907.

In 1911 Horta discovered *M. faerescens*, and in 1914 he found a new Trichophyton, which was subsequently described and named *T. griseum* by Vasconcelos.

Argentina.—In 1907 Urburu discovered *M. fulvum*, and in 1909 *T. orizacatum* and *T. polygonum*.

Central America.—In 1913 Brumpt named a peculiar parasite, discovered by Dr. ...

himself had found a Trichophyton (subsequently named *T. circumlolutum* by Sabouraud in 1909) in white people returning from the Western Sudan.

nigro-circinata

In 1912 he found *T. nodiformans* Castellani in tinea barbae tropicalis and in tinea calvarum.

Tropical Queensland.—In 1914 Priestley discovered *M. scorium*.  
 Prevalence in ...

of cases due to *T. violaceum* Bodin, 1902, var. *khartoumense*.



## TINEA UNGUIUM TROPICALIS

### TINEA UNGUIUM TROPICALIS

**Synonym**—Onychomycosis tropicalis  
Cases of tinea unguium or onychomycosis occur in the tropics and are generally due to the same fungi producing dhobie itch both Epidermophytosis and Trichophytosis. The nails of the fingers as well as of the toes may be affected. Tinea unguium may be caused also by fungi of the genus *Endodermophyton* the nails being often affected in tinea imbricata.

**Symptomatology**—The affected nails have often a peculiar yellowish opaque or blackish discoloration and a rough surface, they become brittle and splitting and chipping of the free border takes place.

The diagnosis is principally based on the microscopic examination of scrapings. A soaking in liq potassae (40 per cent) for twenty four hours is often necessary to disintegrate the nail substance and to find the fungi.

**Treatment**—This is most difficult. The affected nails must be softened by rubbing in liquor potassae and then wet dressings of hypsulphite of soda (25 per cent) or a solution of potassium iodide (grms 5 iodine grm 1 water 1000 c.c.) must be regularly applied. In the onychomycosis found in cases suffering or having suffered from tinea imbricata the daily application of resorcin mixture of benzoin (3i to ʒi) is useful.

### TINEA INBRICATA (TOKELAU)

**Definition**—The term tinea imbricata is used to denote a tropical dermatomycosis or more correctly a group of dermatomycoses due to fungi of the genus *Endodermophyton* Castellanii in clinically characterized by the presence of extensive flaky scaly patches the scales being large tissue paper like firmly adherent by their bases and arranged in concentric rings or parallel lines.

**Synonyms**—As is the case with several other tropical diseases such as leishmaniasis and Oriental sore there is a very large number of synonyms which may be classed as follows—  
(a) From the name of the centres where the disease is first recorded the term Tokelan generally used by French writers, instance the name of an island Tokelau where the malady is common. Other synonyms are Tokelau ringworm used by Tilbury Fox Bowditch ringworm the name Bowditch being by some writers to indicate the island of Tokelau.  
(b) From the name of the patient who first introduced the disease in certain countries. In the island of Tokelau for instance the name of Tunina is Le Pita from Peter the name of Tunina one of the Gilbert Islands who introduced the disease into Tokelau.



used

(d) From the generic name given to the fungus 'Aspergillosis' of Wehmer, 'Lepidophytosis' of Tribondeau, 'Endodermophytosis' of Castellan

(e) From the name of the authors who have more completely studied the disease 'Manson's herpes' 'Turner's herpes,' etc. the term 'herpes' being used by Roux and others in the obsolete meaning of epiphytic skin disease

(f) Terms apparently of unknown origin such as 'Gugo' a denomination much used in the Marshall Islands, 'Cascao' a term used in the Molucca Islands, 'Buckwar,' etc

**History.**—The first recognizable account of the condition is to be found in Dampier's 'Voyage Round the World' published in 1789. Dampier saw the disease in Guam and in the Ladrone century Dentrescasteaux de

Tonga. In 1811 Marsden observed it among the natives of Polo Mas on the west coast of Sumatra. In Alibert's 'Atlas' published in 1832 there is a reference to the disease. In 1841 the disease was recognized by the medical officers attached to the United States Exploratory Expedition led by Commodore Wilkes and Fox in 1844 described it under the name of 'gune' the term used by the Gilbert Island natives

In the reports of the Samoa Medical Mission for the year 1869 there is a good description of the malady by Geo Turner. In 1874 Tilbury Fox gave a description of the complaint under the term 'Tokelau ringworm' sent to him from very similar to drawings given to have been a contamination

authorities who however had no personal experience of it

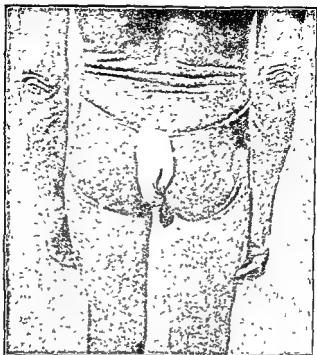
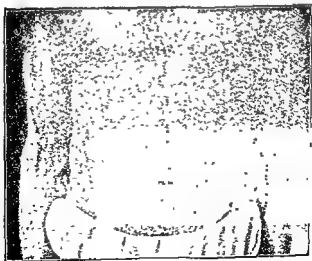
important. He gave a complete clinical description of the malady, and introduced the very appropriate name of *tinea imbricata*, moreover he very correctly described the microscopical appearances



FIG 809.—TINEA IMBRICATA

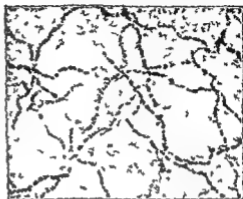
demonstrated that the aspergillus like fungi described by a number of authorities are merely saprophytes and that the true aetiological agents are those fungi for which he has created the genus *Endodermophyton*.

**Climatology.**—The home of *tinea imbricata* seems to have been the Malay Peninsula from whence it spread towards the south and the east to many islands of the South Pacific, northwards to some

FIG. 810.—*TINIA IMBRICATA*.FIG. 811.—*TINEA IMBRICATA*

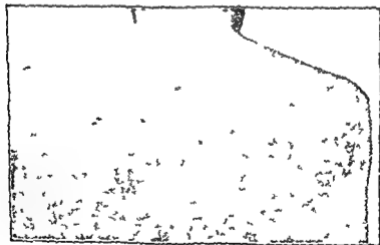
parts of China as far as Foochow and Formosa and westwards to Burma and Ceylon. The Gilbert group of islands seems to have

been infected since the beginning of last century. In 1851 it is said that a native of Tumana in island of the Gilbert group was affected with the disease by a child of a Britisher on island of Tokelau in 1851. In that year it was the disease spread rapidly all over the British Territory of Tokelau. The Tumana man who brought the disease was the father hence the disease became known in British Territory of Tokelau as Le Piti viz. The Father. From Tokelau the disease spread to Samoa according to Turner and Hanger in



TOKELAU AND CALFA  
(From a stained paper of )

1851 and to many other islands where it became known as Le Kelau. At the present time the disease is extremely common in the Malay Peninsula some parts of India and Southern



THE MALAY PENINSULA

1851 and to many other islands where it became known as Le Kelau. At the present time the disease is extremely common in the Malay Peninsula some parts of India and Southern



FIG 810—TINEA IMBRICATA



FIG 811 TINEA IMBRICATA

parts of China as far as Foochow and Formosa and westwards to Burma and Ceylon. The Gilbert group of islands seems to have become heavily infected since the beginning of last century.

In 1853 it is said that a native of Timana in island of the Gilbert group affected with the malarial fever landed at Bowditch an island called also Tokelau in 1859. From that year onwards the disease spread rapidly all over the Bowditch or Tokelau Island. The Timanian man who brought the disease was called Peter hence the disease became known in Bowditch or Tokelau as *Te Peter* (from Tokelau the disease spread to Samoa and other islands in the

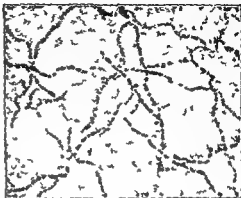


FIG. 1. Distribution of *Tinea imbricata* in the Gilbert Islands.

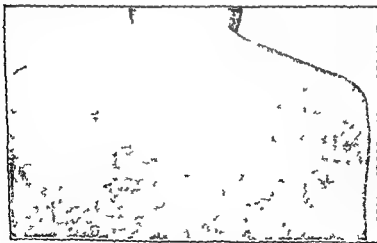


FIG. 2. Distribution of *Tinea imbricata* in the Malay Peninsula and the East Indies.

1860) and to many other islands where it became known as Tokelau. At the present time it is extremely common in the Malay Peninsula and some parts of India-China and Ceylon.

China Borneo Samoa Java the Solomon Islands New Guinea Sumatra Fiji According to Daniels the disease was first introduced into Fiji by some Solomon Islanders in 1870 and within the following two years became *extremely prevalent*. In certain of the Pacific Islands one third to one half the population is affected. The disease is common in some districts of the Philippine Islands the Ladrones the Loyalty Islands New Caledonia and some districts of Burma. Until 1904 the disease was believed to be non-existent in Ceylon but in that year Castellani recorded the first case. During the last few years the disease has greatly spread in this island and it is now fairly common though not so common as in the Malay Peninsula or Fiji. India is said to be



FIG 814—*TINEA IMBRICATA*

so far immune but two typical cases hailing from Southern India have been seen by Castellani. Cases have been reported from Brazil and other parts of tropical America but some doubt has been expressed as to their being cases of true *tinea imbricata*. The cases so far reported from Africa are also doubtful.

The climatic conditions favourable to the rapid development and spread of the disease are represented by a warm damp equable climate with a temperature of 80° to 90° F. the same climate as

have a cold and cool season—such as many parts of India and China—the disease apparently does not spread.

**Ætiology**—The ætiology of this dermatomycosis has been the subject of numerous controversies. Manson first in 1872 described a trichophyton like organism in the squamæ with the laboratory technique of that time attempts at cultivation did not succeed. Blanchard considered it non-<sup>?</sup>

*Trichophyton* " "

stated that  
the squamæ  
His results w  
has been tha  
tribordera  
aspergillus

genus *Lepidophyton*



FIG. 815.—TINEA IMBRICATA

(See scale & hair—plant) Wehrer has described it as a true aspergillus—*Aspergillus tokelau*. The investigations carried out by Castellani have demonstrated that the aspergillus and aspergillus (the fungus) have nothing to do with the disease. When they are present in the squamæ they are merely saprophytes or parasites. By using a special technique the true fungus

is probably more of a cause of the disease than the true fungus. It is found in the hair and causes — as regards age many authorities state that



however, the condition is rare or absent in infants and children, while the persons affected are generally young adults, but it may be found also in very old persons. Women are attacked less frequently than men. Villagers and people living in the country are much more liable to contract the malady than people living in large towns. It is doubtful whether there is any racial disposition. In Fiji, however, it has been observed that while extremely common in the indigenous population it is comparatively rare among the immigrant Indian coolies. The disease rarely, and is due to the habit they

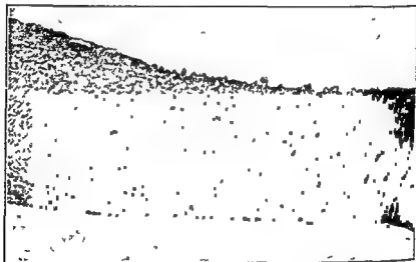
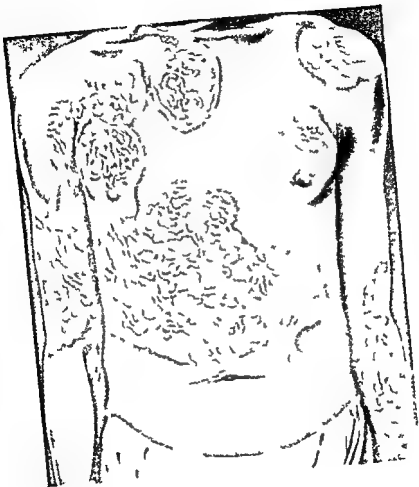


FIG 816.—TINEA IMBRICATA OF THE FOREARM (OLD CASE)

a habit not shared by the Fijians. A hot moist equable climate seems to be the most suitable for the development of the fungus and the spreading of the disease. Manson has justly remarked that the climate which is suitable for the growth of coconuts is

veral small,  
ry itching  
at 6-10-12 large

breaks, and a second scaly ring is formed which extends peripherally inside the first ring, and so on until a very large roundish patch is formed, containing several concentric scaly rings. Manson has aptly compared this formation of rings to concentric ripples



TINIA IMBRICATA

**Differential Diagnosis—Ringworm**—*Tinea imbricata* has an absolutely different clinical aspect from any type of body ringworm, inflammatory signs are totally absent, and the scales are very large flaky, firmly attached by their bases, and arranged in parallel lines or concentric circles. The scales contain an enormous amount of the fungus.

*Ichthyosis*—The medical man newly arrived in the tropics often mistakes the disease—when of the diffuse type—for ichthyosis, so much so that it has also received the name of tropical ichthyosis. The microscopical examination of the scales will clear the diagnosis at once.

*Pityriasis rubra*—In *tinea imbricata* there is not the intense hyperæmia of the skin, and the scales are firmly attached. The microscopical examination will clear the diagnosis in any doubtful case.

*Tinea intersecta*—*Tinea intersecta* begins in a manner somewhat similar to *tinea imbricata* dark-brownish patches being present at first, and the fungus in both eruptions growing between the superficial and deep strata of the epidermis. In contrast to *tinea imbricata*, however, the eruption never develops in concentric rings, the scales are not firmly attached, and the cure is easy.

**Prognosis.**—The disease has no tendency to spontaneous cure, and the treatment is difficult. The general health is not much affected, but the patient complains of the disfigurement, which is very great, and of the pruritus, which in the hot season may be unbearable. Europeans complain also of pain especially if the fungus attacks the hands. In very chronic cases signs of anæmia,

• • • • • Coolies affected with  
 • • • • • to work owing to  
 • • • • • of great economical  
 • • • • • apply of labour on  
 estates etc.

**Treatment.**—Every medical man practising in the tropics well knows how difficult is the treatment of *tinea imbricata*. It is easy to obtain a temporary improvement, and even a disappearance of the eruption, but as soon as the treatment is discontinued the

by Manson, or resorcin  
 31, tr benz co, 31, 11  
 an ointment (5 per cent)

In the Colombo Clinic of Tropical Medicine one of us made various experiments to test the efficacy of the various medicaments by applying

appear  
 not per  
 typical

scales reappear

# TINEA IMBRICATA (TOKELAU)

Some and other ointments of mercurial preparations do not induce any improvement in the eruption  
Hydrolyol and Naphthol ointments may cause a slight improvement.  
Salicylic Acid and Episcasin ointments have no effect whatever  
Zinc ointment (20 to 50 per cent) may induce a temporary improvement

Formalin is very effective for localized patches. The usual 40 per cent solution is applied with care treating each time a small portion of the eruption which is best relieved by applications of iced water. Soon after the application of formalin the patches become dark brownish which colour lasts for a few days when they clear. Care must be taken not to apply the formalin to too large portions of the skin and not to repeat the application too often otherwise a peculiar form of pigmentation similar to leucodermic patches may appear later on to which disfigurement coloured patients strongly object.

**Chrysoarobin**—The repeated application of chrysoarobin ointment (30 grains to 1 ounce of vaseline) may induce a strikingly rapid improvement in cases which are not of long standing. The eruption however recommences a few days or weeks after its apparent disappearance. Chrysoarobin is a very toxic medication the patient must be watched and the urine regularly examined. In one of our cases symptoms of absorption appeared after a single application.

**Salicylic Acid and Methyl Salicylate** have very little if any action on the fungus.  
**Tinctura Iodi and Lintimentum Iodi**—Tinctura Iodi freely applied induces a very marked improvement which however is not permanent. Strong iodine liniment as recommended by Manson is most effective it cannot be used freely however on patients with a delicate skin such as women and children.

**Resorcin and Tincture of Benzoin**—Resorcin alone or mixed with salicylic acid in alcoholic solution and in ointments has very little efficacy. If however resorcin be dissolved in tincture benzoini composita (60 to 120 grains of resorcin to 1 ounce of tincture of benzoin) very good results are obtained. It is now the routine treatment for tinea imbricata in the Colombo Clinic. It is to be noted that the resorcin dissolved in tincture of benzoin little attention on the eruption. The treatment must be continued for several hours be applied freely once or twice daily on the affected regions. If the sole body be affected one day one half is painted and the other days the other half alternately. The treatment must be continued for several weeks. Once or twice a week the patient is given a very hot bath and scrubbed all over with sand soap. Symptoms of absorption are rare it is always prudent however to proceed at first with care as it is well known that in individuals may be met with though rarely showing idiosyncrasy to resorcin.

**Prophylaxis** Some authorities recommend isolation this is good wherever possible but in regions where the disease is or has become endemic usually the great number of people suffering from the affection render the measure hardly practicable. In those tropical countries however where the disease has not yet appeared medical officers would do well to be on the look out for it and if a case is reported the patient should certainly be kept isolated thoroughly treated before being allowed to mix with the general population and all infected clothing should be boiled or steamed. We have seen an epidemic of tinea imbricata in a hospital in a patient suffering from the disease was admitted and allowed to mix with the other patients. There is a general native belief

anointing the body with coconut oil or other oil will prevent infection there may be some truth in the belief but such a measure cannot be carried out in Europeans Any itchy scaly spot in the slightest way suspicious of incipient tinea imbricata should be immediately treated with lin iodii chrysarobin ointment or resorcin dissolved in tr benzoni While the treatment of tinea imbricata in an advanced stage is extremely difficult it is easy to stop the initial patches by these means

### TINEA INTERSECTA (vide Plate XI)

This dermatomycosis and its fungus were first described by Castellani in 1907 It occurs in Ceylon and Southern India

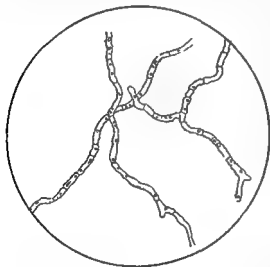


FIG 820—FUNGUS OF TINEA INTERSECTA  
(From a scale in liq potassæ)

**Ætiology**—If a portion of one of the brown patches or a scale be removed and treated with liquor potassæ the fungus is easily detected The fungus (*Endodermophyton castellani* Perry 1907) grows between the superficial and the deep strata of the epidermis It is present on the inner surface of the scales but not on the external surface A very remarkable fact is the extreme rarity of free spores in fact in several cases one does not succeed in finding spores The mycelium is fairly

imbricata  
which are  
and  $3\frac{1}{2} \mu$

Each segment presents in fresh preparation two refractile bodies one at each extremity No aspergillar fructifications nor clusters

spots are at first slightly elevated and dotted often with minute dark papules. The patches are dark brown in colour much darker

PLATE XI



*TINEA INTERSECTA* F. BRADY



## TINEA FLAVA

surrounding skin and presents a smooth tense surface at a time the surface of the patches is no longer tense. After a while the surface of the patches is no longer tense, it becomes what shrivelled and dry, superficial cracks appear in it so cracks become deeper intersecting the brown surface. Later up scales whitish intersecting the brown surface. Later the scales are often removed by friction and whitish roundish patches only remain. The eruption never develops in concentric rings like tinea imbricata the patches remain isolated or fuse together, forming irregular larger patches. Some patches may disappear spontaneously after a time. The general health of the patient does not seem to be affected. In some patients there is a slight degree of eosinophilia.

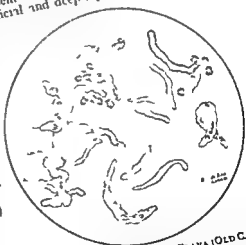
**Diagnosis** — When the eruption is in the very first stage it might be mistaken for a form of pityriasis versicolor. In pityriasis versicolor, however the epidermis does not split moreover in tinea intersecta the fungus is not found on the surface it grows between the superficial and deep layers of the epidermis. Tinea imbricata begins in a manner somewhat similar to tinea intersecta dark brownish patches being present and the fungus in both eruptions growing between the superficial and deep layers of the epidermis. In contrast to tinea imbricata however the eruption of tinea intersecta never develops in concentric rings is far less severe patches may heal spontaneously and is cured without much difficulty.

**Treatment** — Tincture of iodine and the usual antiseptic ointments such as chrysotholbin (2 to 5 per cent) answer well.

## TINEA FLAVA (*vide* Plate XII)

**Synonyms** — Tropical Pityriasis Versicolor of the old authors Microsporosis (Jeanselme) Pityriasis Squamosa (Crocker)

**FIG 821** — FUNGUS OF TINEA FLAVA (OLD CASE)  
(From a specimen stained by the Norman-Walker method)



Flava (Castellani) Achromie Parasitica  
Versicolor Flava (Castellani) Achromie Parasitica



This dermatomycosis is extremely common in all tropical countries and especially so in Southern India Ceylon Malaya Java Indo China and China By many authors it has been and is still confused with the ordinary pityriasis versicolor of temperate zones the investigations of Castellani and Jeanselme, however have clearly proved that it is a distinct entity

109



FIG 822.—TINEA FLAVA ON THE FACE OF A SINHALESE

swellings constrictions and other irregularities in their shape The spores are roundish or oval ( $3.50$  to  $4.50 \mu$ ) and have a double contour In recent cases the fungus is abundant with plenty of mycelium and spores which often run into clusters In old chronic patches the fungus becomes very scanty, the spores are not numerous very few or absent In the latter case the patches are very small and the fungus is not abundant In the former case the patches are very large and the fungus is abundant of the fungus see p 1099

**Symptomatology**—The affected parts are yellowish of much lighter colour than the surrounding healthy skin the yellow colour



FIG 823—TINEA FLAVA ON THE BACK

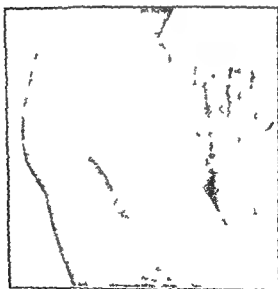


FIG 824—TINEA FLAVA OF THE ARM

may be of various tinges, from dark deep orange yellow in some cases to very light canary yellow in others. The patches are of

This dermatomycosis is extremely common in all tropical countries and especially so in Southern India Ceylon Malaya Java Indo China and China By many authors it has been and is still confused with the ordinary pityriasis vesicolor of temperate zones the investigations of Castellani and Jeanselme however have clearly proved that it is a different affection

**Ætiology**—The affection is due to *Malassezia tropica* Castellani 1905 The mycelial threads are generally thick with numerous

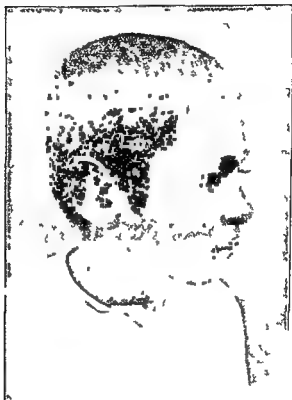


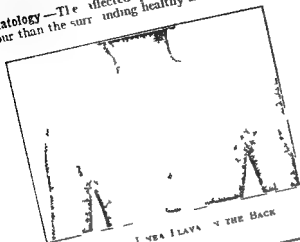
FIG 872.—TINEA FLAVA ON THE FACE OF A

swellings constrictions and other irregu! t

very scanty and is even more irregular in shape t  
 patches (degeneration forms of the fungus) A  
 of the fungus see p 1099

**TINIA FLIVA**

symptomatology—The affected parts are yellowish of much  
 deeper colour than the surrounding healthy skin the yellow colour



—TINEA FLAVA ON THE BACK

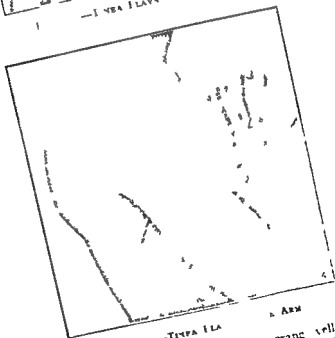


FIG 324—TINEA FLA

ARM

of various tinges from  
 very light canary yellow to

orange yellow or  
 175. The patches

peculiar parchment-like feeling of scleroderma. The microscopical

taken for a form of *Seborrhœa corporis*. The microscopical examination will clear the diagnosis, no *Malassezia* fungus being found in *seborrhœa corporis*.

**Treatment.**—*Tinea flava* shows no tendency to spontaneous cure unless the patient moves to a cold climate. Even then, very often the cure is only apparent, as the condition reappears during the hot weather. The treatment is difficult. Turpentine applied daily, followed by a naphthol or epicarim ointment (2 to 5 per cent), or a salicylic-resorcin ointment (resorcin 3i, acid salicyli gr x, vaselin 3i), is often successful, but the treatment must be continued for months. On covered parts of the body tincture of iodine may be used, or a chrysarobin ointment (2 per cent.).

They become again normally pigmented.

### TINEA NIGRA.

**Synonyms.**—Pityriasis Nigra (Castellani), Microsporosis Nigra (Castellani)

This affection is fairly common in India, Ceylon, Java Federated Malay States, and China. The first account of this, or a very similar, dermatomycosis was published by Manson in China in 1872. Manson's observations, however, were forgotten, as they were not quoted by him in his subsequent works. Castellani, in 1905, redescribed the disease in Ceylon, and succeeded in growing the fungus.

**Ætiology.**—The affection is caused by a fungus of the genus *Cladosporium*—*C. mansoni* Castellani, 1905. The fungus is found very abundantly in the lesions, the mycelial elements are rather short, 18 to 20  $\mu$  in length, and 2 to 3  $\mu$  in diameter. Sometimes

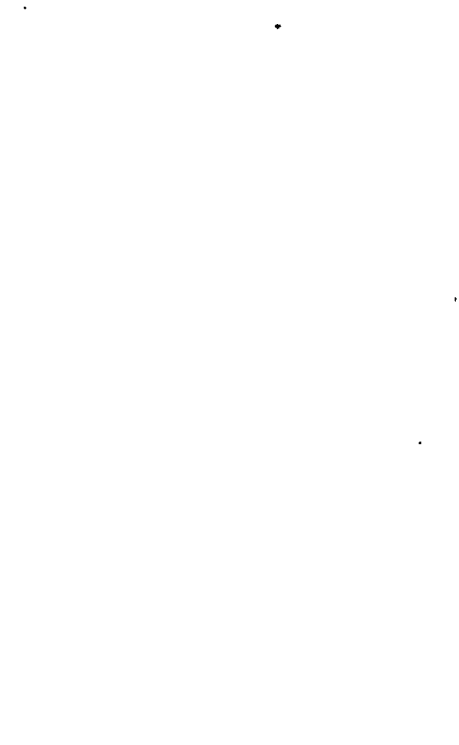
The spores  
 u They are frequently arranged in clusters

The fungus is easily cultivated by inoculating scrapings of the affected patches on maltose agar. After two to four days roundish hemispheric colonies appear, which are black, but at first have usually a greenish tinge, and may present at the periphery some radiating, delicate, pale greenish hyphæ.

PLATE XII



TINPA FLAVA ON THE FACE AND  
TINPA NIGRA ON THE NECK



slow and takes place at the bottom of the tubes with formation of a black or greenish black sediment

The optimum temperature for the growth of the fungus is between 30° and 32° C above 35° C and under 25° C the growth is much slower and may be nil under 20° C Further details on the fungus may be found on p 1100

**Symptomatology.**—The affected parts are of a black dull lustreless colour much darker than the surrounding dark healthy skin of the native The patches may be small roundish and separated from one another or may coalesce the patches are often slightly elevated and may present a slight desquamation Little if any pruritus is present The face is not usually affected though the eruption may be found on practically any other region of the body The neck and upper portion of the chest are apparently the regions most frequently affected *Tinca nigra* usually attacks natives We have however

seen it also in one of our European patients who went for a pleasure trip to Burma where he remained about a month On coming back to Ceylon he noticed a small roundish very slightly elevated non desquamating black patch on the palm of his left hand There was no pruritus The patch spread slowly for two months reaching the size of a sixpenny piece It disappeared after a single application of formalin three months later it re-

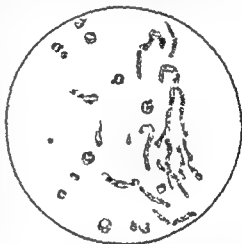


FIG 827.—FUNGUS OF *TINCA NIGRA*

appeared in the same place as a tiny black dot which slowly spread Another application of formalin caused it to again disappear From the patch a fungus was grown identical with the one found in native cases

**Mixed Infections.**—A mixed infection of *tinea nigra* and *tinea flava* is somewhat frequently met with Several of our patients had on the neck a few round patches of *tinea nigra* and on the face some smooth yellow round patches of

*tinea flava* and the fungus (*Massaria versicolor* or) is morphologically very different and cannot be grown In *chloasma bronzium* no fungus is found



**Treatment**—The disease is easily curable except when it attacks the palms of the hands where the treatment must be more prolonged. A salicylic alcoholic lotion (2 per cent) followed by a resorcin ointment (resorcin 31 vaseline 31) answers well. When the patches are small pure formalin may be used with care.

### ERYTHRASMA

This affection is frequently met with in the tropics and is common on the continent of Europe though apparently rare in America.

**Ætiology**—It is caused by a fungus discovered by Burchardt in 1859—*Nocardia minutissima* Burchardt. This hyphomycete is very delicate and slender less than  $1\ \mu$  in breadth is found in the superficial horny layer of the affected parts. Ducrey and Reale claim to have succeeded in cultivating it but their results have not been confirmed. The description of the fungus is found on p 1061.



**Symptomatology**—The eruption generally affects the genito-crural and axillary regions but may occasionally spread to other parts of the body. It is characterized by the presence of brownish reddish patches rounded or irregularly shaped but with well defined borders. The borders are not elevated the surface of the patches appears slightly furfuraceous and has often a somewhat greasy feeling. There are no subjective symptoms except occa-

is easily  
versicolor  
nt by the  
the micro  
f *Nocardia*

*minutissima* Burchardt a fungus morphologically very different from *Malassezia furfur* Robin or *M. tropica* Castellani. The differential diagnosis from tinea cruris has been discussed in this chapter under the heading Tinea Cruris (p 2042).

As first noticed by Manson after tinea cruris has been cured the genito-crural region may in some cases present for years a peculiar brownish discoloration and be slightly furfuraceous—a condition

re-

eri

water, carbolic (1r, or sand soap, and then regularly applying a resorcin-salicylic ointment (resorcin, gr. x to xxx, ac salicyl, gr. x to xx; vaseline ℥i), will soon cause the eruption to disappear. Instead of the ointment, a hyposulphite of soda (℥ drachm to 1 ounce) or sulphurous acid lotion may be used.

### BLASTOMYCOSIS.

**Synonyms.**—*Saccharomycosis Hominis*, *Oidiomycosis Dermatitis Blastomycetica*, *Blastomycetic Dermatitis*, *Zymonematosi*.

**Definition.**—The term blastomycosis covers a group of closely allied pathological conditions due to fungi of the genera *Saccharomyces*, *Cryptococcus*, *Coccidioides*, *Oidium* and *Monilia* generally characterized by the presence of warty patches and minute epidermal abscesses.

#### Historical and Geographical.

—Wirkke, in 1890 described in Buenos Ayres two cases of papillomatous eruption in which he found peculiar bodies which were at first considered to be protozoa; hence the disease was called protozoic dermatitis. Later Gilchrist and Ophuley showed them to be vegetable parasites. Gilchrist in 1894 described yeast-like organisms in sections taken from a scrofuloderma-like eruption. In the same year independently Husse and Buschke published a case of a pyramidal condition due to a *Cryptococcus*. The disease has been investigated chiefly



FIG. 519.—BLASTOMYCOSIS OF LEG.

by American observers among whom Ricketts, Ormsby, Hyde, Montgomery and Pusey may be mentioned. The malady occurs

inducing identical clinical conditions. In the tissues all the organisms exist as yeast like oval or roundish cells.

Among the organisms which cause the disease Ricketts distinguished four types —

1 Blastomycetoid or yeast like type reproduction by budding in cultures only oval or roundish cells are seen while mycelial tubes are as a rule absent.

2 Cryptococcus like type reproduction by endosporulation within the tissues.

3 Endomyces like type the cultures present abundant submerged mycelium which breaks up into chains of endoconidia proliferation by budding is rare.

4 Hyphomycetoid type cultures present aerial hyphæ and submerged mycelium proliferation by gemmation occasionally seen. There are many transition forms between these four groups.



FIG 830 —BLASTOMYCOSIS OF THE UPPER LIP ULKERATIVE STAGE

(From a case in the Colombo Clinic)



FIG 831 —BLASTOMYCOSIS OF THE MOUTH

(From a photograph by Splendore)

**Histopathology** —There is marked proliferation of the epithelial layers with elongated irregularly shaped downgrowths into the corium and epithelial globi are seen. The cells of the rete are swollen and there is between the cells a polymorphonuclear leucocytic infiltration. Here and there minute milium abscesses are present. In leucocytes are containing the organism is mostly found. The corium presents a general cellular infiltration made up of polymorphonuclear leucocytes and young connective tissue cells. The vessels are dilated and their walls thickened. Splendore has noted that when the lymphatic glands

are affected which is of rare occurrence they may present histologically a tubercular appearance

**Symptomatology**—The disease as seen by us in Ceylon is characterized by the presence of elevated warty patches showing especially at their margins minute epidermal abscesses and often small ulcers covered by yellowish crusts. The eruption may be gyrate. The lesions at a later stage may become more deeply ulcerated the process of ulceration generally beginning at their centre. There is very little or no pain and very slight or no pruritus. The lesions may heal spontaneously leaving as a rule soft smooth scars. Occasionally the affection recommences after a time in the scar. The lymphatic glands are not often involved. In some cases subcutaneous abscesses and gummas



FIG. 832.—BLASTOMYCOTIC ULCER  
(From a photograph by Splendore)



FIG. 833.—BLASTOMYCOSIS GLUTEALIS  
(From a case in the Colombo Clinic)

like swellings may be formed. All regions of the body may be affected. The course is very chronic the disease lasting often for many years.

**Clinical Varieties.**—Several varieties can be distinguished —

- 1 The common cutaneous type or North American and Asiatic type
- 2 The oro pharyngeal blastomycosis
- 3 The Coccidioides blastomycosis
- 4 The gluteal blastomycosis

1 *Common Cutaneous Type*—The description we have given of the disease refers to this type which is quite common in Ceylon Southern India Philippine Islands Indo China Tonkin and probably in many other parts of the tropics. A very frequent localization in Ceylon is the upper lip (see Fig 830) the disease extending later occasionally to the nasal mucosa and very rarely to the oral mucosa. In several of our cases a monilia like fungus was grown.

2 *Oral Blastomycosis*—This variety has been investigated by Lutz and Splendore in South America. Splendore considers the

..

or frambesiform patches which later may undergo deep ulceration. The disease later invades the pharynx nose larynx and bronch,

ease) was

Posadas

It was

at first considered to be of protozoal origin. The lesions on the

very deep and connected with each other but in our cases did not communicate with the intestine. The pus does not contain grains as is the case with actinomycosis. The patient may complain of pain and discomfort on sitting down. The disease is chronic.

Maxwell has reported from Formosa cases of a fistulous disease of the buttocks, which may be of the same nature. He is inclined however to consider them to be of amoebic origin.

## BLASTOMYCOSIS

**Diagnosis**—This is based in the usual type of the malady on the presence of verrucose patches with micro-abscesses in which the fungi are found. The disease has been often confused with tuberculousis verrucosa with a syphide with an epithelomatous lesion and in the tropics with atypical frambesia and even ringworm. The microscopic examination and cultivation of scrapings will better of the pus of the milary abscesses present in the lesions will be necessary to clear the diagnosis. A droplet of the pus or a minute portion of teased tissue is placed on a slide with a drop of a 30 per cent solution of potassium hydrate and a cover glass is placed on the preparation after about half an hour in a temperate climate and generally a few minutes only in a tropical climate the tissue and pus cells are disintegrated by the potash solution while the organisms being resistant can be easily seen. Cultures should also be made from the pus. It is important to note that yeast



834 — FUNGUS FOUND IN DERMATOSIS HYPHOMYCETICA INDICA (BROTH CULTURE)

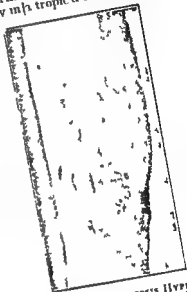


FIG. 515 — DERMATOSIS HYPHOMYCETICA INDICA (P. 1936)

organisms may be frequently found as saprophytes on the surface of various ulcerated skin lesions which have nothing to do with true blastomycosis. Oral blastomycosis may occasionally be confused with espumosa in which it is differentiated by the absence of Leishmania and the presence of bodies containing numerous spores as in the case of endospores. *Gluteal blastomycosis* is distinguished from the mucic and mycetomatous conditions by the absence of the characteristic characters of the fungi from a syphilitic condition. The similarity of a mercurial treatment, from tubercular lesions

disease by the massive diffuse induration, and absence of tubercular cutireaction

**Prognosis.**—The disease very rarely heals spontaneously. The general health in the common type of the malady is not much affected but the patients complain of the disfigurement. Occasionally the organisms from the skin lesions enter the general circulation, and a condition similar to pyæmia develops. Cases of systemic blastomycosis terminating fatally, without any skin lesion have also been described. The prognosis of blastomycosis coccioides and oral blastomycosis is bad, while blastomycosis glutealis is most persistent though the general health is not much affected.

**Treatment.**—Potassium iodide given in large doses (gr. xv. to xx three or four times daily), has a beneficial effect, though it is not so efficacious as in sporotrichosis. Disinfectants to the lesions is useful.

Disinfectants—e.g. tincture of iodine, etc. may bring about a cure. The following ointment is useful, especially in the localization to the upper lip: Ichthyol, gr. xv, unguentum belladonnæ ʒi, vaselinum ad ʒi. No treatment is apparently of much use in blastomycosis coccioides, in oropharyngeal blastomycosis or in blastomycosis glutealis.

*Dermatosis Hyphomycetica Indica*—This term has been used by Castellani to indicate a peculiar hyphomycetic condition he has once seen in Ceylon. The patient had a number of gummatous swellings and indurated patches, but no warty lesions were present anywhere. A fungus was isolated which in various sugar broths and ordinary broth produced very long filaments but owing to an accident could not be further studied.

### SPOROTRICHOSIS.

Schenk, in 1898, described a case of multiple chronic abscesses in the pus of which a *Sporotrichum* was found. Heiktoen and Perkins reported two similar cases also in the United States in 1900. De Beurmann published in 1903 a case of similar nature in France. De Beurmann and Gougerot, from 1906 onwards, have published many cases, and have completely investigated the subject of human sporotrichosis bacteriologically and histologically, as well as clinically. Their researches have been confirmed by Gaucher and Monier-Vinard, Duval and Fago, Vaquez, Bonnot, Lambry, Adamson, Esmen, and many others. Cases have been reported from the tropics by Lutz and Splendore in Brazil, and by us in Ceylon. Clair has observed the disease in Arab stokers on board

to the genus  
been so far

(*S. schenki* Hektoen and Perkins 1901)

For the description of these organisms see chapters on Fungi (p. 2117)

The species so far found in the tropics are—*S. leuromanni* Matruchot and Ramond in Brazil and Africa *S. asteroides* Splendore 1909 in Brazil and *S. indicum* Castellani 1908 in Ceylon. These fungi are morphologically very similar. In the human lesions the fungus appears morphologically as a yeast and is very scarce. In cultures mycelial threads and numerous spores are seen. The spores are ovoid 5 to 6  $\mu$  in length by 3 to 4  $\mu$  in breadth. The mycelial filaments are very slender (2  $\mu$ ) in *S. leuromanni* and *S. schenki*,



FIG. 836 SPOROTRICHIC LYMPHATIC VESSEL

(From a photograph by Splendore)

somewhat broader (2½ to 3  $\mu$ ) in *S. indicum*. *S. asteroides* is characterized by the presence of peculiar radiate bodies in the affected tissues. These fungi grow easily on the ordinary culture media, best of all on Sabouraud's peptone glucose agar. Colonies develop from the fourth to the tenth day as small white points surrounded by a delicately rayed aureola of whitish colour. They

will still develop in place by contact with unclean vegetables.



The fungi may also apparently, live saprophytically in the oral cavity

mo

intraperitoneal inoculation The rat is the most susceptible animal In it Lutz and Splendore have described a spontaneous sporotrichosis due to a *Sporotrichum* apparently identical with *S. beurmanni* Moore and the bite of a field mouse

well *S. schenki* and *S. bei*

observed to occur spontaneously in the dog (Gougerot and Caravan) and in the mule (Fontoynt and Carougeau) in Madagascar

**Histopathology.**—The histopathology of the cases due to *S. beurmanni* has been investigated by De Beurmann and Gougerot, that of the cases due to *S. asteroides* by Splendore, that of the cases due to *S. indicum* by ourselves Whatever the causative species of *Sporotrichum* the histological lesions are apparently the same and correspond to the three principal types described by De Beurmann and Gougerot—viz (1) An epithelioid type with presence of giant cells—this corresponds to the tubercloid type of De Beurmann and Gougerot, (2) a lympho connective tissue or syphilitic reaction, (3) a polymorphonuclear or ecthy-matiform type

**Symptomatology.**—In a well marked case several gummatous like swellings situated in the subcutaneous tissue are present on various parts of the body—the arms legs and trunk The size

On palpation  
the skin becomes  
tense From the

fistulous opening a yellowish homogeneous pus is slowly evacuated or at times a thin serous discharge In some cases the suppuration ceases granulation sets in and a coarse cheloid like scar remains In other cases the fistulous opening enlarges and a crateriform ulcer with often a fungating fundus develops The lymphatic glands may occasionally become affected The course of the disease is very chronic In some cases deep gummata develop under the

36 granulating  
The general

...

th are —  
nd ascending

sporotrichic lymphangitis

2 The disseminated gummatous type

3 The disseminated ulcerative type presenting often polymorphic lesions—viz syphilitic like tubercular like ecthy-matous rupial furuncular

4 The extracutaneous type with sporotrichic lesions of the mucous membranes the muscles, the articulations the bones the organs of special sense, the internal organs—lungs kidneys etc.

### ACLADIOSIS

De Beurmann has put on record a case of mycetoma of sporotrichic origin. Cases of systemic sporotrichosis have been described

**Diagnosis**—The principal clinical signs on which to base a probable diagnosis of sporotrichosis are the presence of gumma like lesions while the patient is in a good general state of health, the mixture of the nodes of different appearance partial cup-shaped softening of the nodes breaking down in the centre and ending in ulceration with violaceous edges generally undermined, presence of viscous pus or of a serous lemon yellow discharge, indolent evolution absence in most cases of enlarged glands. The definite diagnosis can only be made by bacteriological methods. The simple microscopical examination of the pus of the abscesses or scrapings of the ulcers is not sufficient as the fungus is extremely scarce. Cultivation must be resorted to. A few glucose-agar tubes are inoculated and kept at room temperature without capping. After four to ten days the first colonies of *Sporotrichum* will appear.

**Treatment**—Potassium or sodium iodides in full doses (15 to 20 grains three to four times daily) well diluted in water or milk induce a rapid disappearance of all the lesions. In persons who cannot take potassium iodide salicin in the same dose may be given in cachets. The ulcerated lesions may be dressed with a solution of potassium iodide 20 parts iodine 1 part water 500 parts. Surgical measures should be avoided.

Pinoys has noted that the action of the iodides is increased by a salt free diet.

**Prophylaxis**—Any small wound should be disinfected with tr. iod.

### ACLADIOSIS

**Definition**—An ulcerative dermatomycosis caused by *Acladium castellanii* Pinoy.

**Historical and Geographical Distribution**—The condition has been observed by Castellani since 1917 in Ceylon but he did not fully describe it until 1916. Cases have been observed in Ceylon the Federated Malay States and Malacouira.

**Etiology**—The condition is caused by a fungus which Castellani isolated in Ceylon. Cultures were sent to Professor Pinoy of the Pasteur Institute who investigated it bacteriologically and classified it giving it the name of *Acladium castellanii* Pinoy, 1916. Professor Pinoy's description may be quoted—

The growth on artificial media (such as carrot potato glucose agar) consists of many small roundish masses which later on may coalesce covered by spiculated formations giving them a prickly appearance and consisting of erect, straight filaments parallel to each other or at times interlacing. The filaments are approximately 2  $\mu$  long in diameter and carry laterally pseudocilia of variable shape cylindrical pyriform or spherical attenuated at their points of insertion. Most of these pseudocilia are 4  $\mu$  in size at their points of insertion. This type of fructification resembles the type *Acladium* described by Robin in certain species of the genus *Acladium* become detailed and then develop by spinous formed. Certain filaments produce spores.

chlamydo-spores arranged in small strings, as found in certain fungi of the genus *Fusarium*. These small chains of chlamydo-spores are very frequently terminal the dimensions being variable—8 to 10 microns (Fig 595 p 1113)

In cultures on carrot and potato the colonies are white, on glucose agar often amber colour. Very old cultures may show a certain

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clear type (ecthy-matous)

**Clinical Symptoms.**—In a well-marked case ulcerative lesions are present all over the body, though they are in smaller number or absent altogether on the face scalp palms, and soles. Most of the ulcers are sharply defined, roundish or oval, with red granulating fundus. Their appearance is well shown in the illustration a photograph of a Ceylon case. In many cases there is abundant purulent secretion, which collects and dries up in thick yellow crusts



FIGS 837 AND 838—*ACLADIUM CASTELLANII* PINGYI HANGING DROP CULTURE

(a) After twenty four hours (b) after three days growth

covering the ulcers. Gummata-like nodules and furuncle-like lesions may be observed. The superficial lymphatic glands may be enlarged. The lesions in most cases give very little pain, or none at all. Itching is often completely absent, but occasionally the patient

negative. In the first two cases red blood-corpuscles and haemoglobin were slightly below the normal, in one there was eosinophilia (5 per cent), with *lumbricoides* infection was examined,

**Diagnosis**—A positive diagnosis can be made with certainty only by cultural methods. The microscopical examination alone is of

isolated colonies  
spheric and often  
may not fuse

to other each colony then remains separate reaches a large size and occasionally presents peculiar radiating furrows as seen in certain pieces of trichopteryx. In many cases where the material has been collected from ulcerated lesions the fungus grows in symbiosis with a cocculus and it may be difficult to separate the two organisms.



FIG. 519.—ULCERATION OF THE ARM

The malarial is not taken for a typhoid condition. The history, the negative examination of the lesions for sporozoites, the failure of mercury and salivary treatment will exclude it. When the lesions are covered by raised, thick, bright yellow crusts the condition must be differentiated from yaws. In accladiosis, on removing the crusts ulcers are found while in yaws the typical firmness nodules will be seen. In scrapings from yaws lesions the treponemes will be found. Accladiosis can be differentiated from sporotrichosis and other affections of lymphoretic origin by cultural methods.

**Prognosis**—The course of the disease may be very long and there is very little or no tendency to spontaneous cure, but if a proper treatment is carried out a cure can be obtained fairly rapidly in the majority of cases. A few cases respond to treatment extremely slowly.

**Treatment**—Pyrarolium sulphuratum in full doses (20 gr. per diem) acts satisfactorily. The drug appears to act at times more rapidly if given according to Professor Pinoy's method—viz. in conjunction

with a salt-free diet. If potassium or sodium iodide is not well borne, salicin and other similar preparations may be tried, but the result is not so satisfactory. Mercury and arsenic have no effect on the course of the malady. As regards local treatment, it is sufficient to keep the ulcers clean by using a weak mercury perchloride lotion.

### CRYPTOCOCCOSIS EPIDERMICA.

by Castellani) was first described found cases in the Sudan and in the Balkanic Zone.

**Ætiology.**—The causal organism is *Cryptococcus epidermidis* Castellani 1914.

**Symptomatology.**—The condition is fairly frequent in Europeans who have resided for some years in the tropics, but is also found in natives. It is characterized by the presence on the arms and more rarely on the chest and neck of small roundish patches of a dirty yellow or brownish colour which can generally be removed by thorough scraping. These patches consist of large numbers of blastomyces like elements of various size rounded or oval which so far have not been cultivated.

**Treatment.**—Thorough scraping with sand-soap and hot water is generally sufficient to remove the patches. In obstinate cases a salicylic sulphur ointment is useful.

### INTERTRIGO SACCHAROMYCETICA

**Synonym.**—Intertrigo Blastomycetica

**Remarks.**—Cases of this affection have been observed by Castellani in Ceylon and similar ones have later been reported by Whitfield and others in Europe. The affection is apparently rare. It generally attacks the scrotocrural and axillary regions. The affected skin is red and there may be slight exudation. The borders of the eruption are fairly well marked but never elevated. In most cases there is not much itching and the affection may recover spontaneously.

**Ætiology.**—In scrapings a *Saccharomyces* (*S. samboni* Castellani 1907) which is easily cultivated on sugar media is found or in other cases fungi of the genus *Monilia*.

**Treatment.**—The treatment consists in washing the affected parts

### ASPERGILLOSIS AND PENICILLIOSIS OF HAIRY PARTS

The affected hairs—generally those of the beard and moustache occasionally of the axilla—present dirty greyish or whitish punctiform formations which on microscopical examination are seen to consist of penicillar or aspergillar fungi (*Penicillium barbæ* Asper

## PINTA

*(his barbæ)* Occasionally both types of fungi are found on the same patient. The affection is very chronic. The diagnosis is easy the characteristic aspergillus and penicillium fructifications distinguishing this condition from other parasitic nodular affections. The simplest method of treatment is by shaving and afterwards using regularly a medicated soap such as carbolic soap, tar soap or sulphur soap. If the patient does not wish to shave his beard turpentine may be tried.

In natives who do not bathe frequently such as old persons and beggars the skin presents often large dark patches due to accumulated dirt in which aspergillus and penicillium fungi are often present in a saprophytally. This condition has nothing to do with pinta as a thorough scrubbing with soap will remove the dirt and the aspergillus and penicillium fungi which may be present.



FIG. 840.—PENICILLIUM FRUCTIFICATION

## PINTA.

**Synonyms**—Mal de los Pintos Mal del Pinto Curate Tinta Jurica Pannus (viticus) (Alibert) Tache Endémique des Cordillères (Alibert) Loma Cutiva Bulpiss (Lerch)

**Definition**—The term pinta does not indicate a single disease but a group of closely allied dermatomycoses characterized by the presence of patches of various colour due to different species of fungi of the genera *Aspergillus*, *Penicillium*, *Monilia* and *Montoyella*.

**History.** The disease first began to draw the attention of medical writers in the eighteenth century though it was apparently well known to the inhabitants of the affected regions since remote times as it is found to have formed the subject of prayers and supplication used by the Aztecs centuries before the Spanish Conquest. A short description of the malady is found in the *Encyclopædia of Pizarro* of Mexico in 1709 and in Juan de Velasco's *Historia* in 1783 in Columbia. Velasco believed the malady to have been imported by Africa in slaves. A fairly complete description is given by Alibert in 1823 under the name of tache endémique des Cordillères or pannus viticeus. Among the modern authors clinical and pathological investigation of Gomez Uribe Uribeel Iriz Ruiz and Santos into the etiology of pinta. More recently the investigation into the etiology of pinta. Montoya has been of the greatest importance.

**Climatology**—Pinta is practically limited to tropical America where it is found in Venezuela Peru Chili Central American Mexico. Cases have been reported from Brazil. It is especially common in Columbia where according to Montoya 4 per cent of the population is affected. There the patient affected. The disease is not reported

tributed; in each country there are localities where the disease is common, while other districts are almost unaffected. In Columbia it is the northern province of Santander which is more particularly affected; in Mexico the disease is most frequently found in the provinces of Tabasco, Chiapas, Valladolid, Michoacan.

A few cases

Goodman,

what simil.

did not find any fungus. A few isolated cases have also been

contained in the waters of the mines and other localities where the malady is endemic, others considered it to be due simply to insanitary conditions, insufficient food and a hot and damp climate, others, again, believed the affection to be induced by the action on the skin of volcanic cinders, while according to some authorities, the malady was an hereditary complaint.

Ruiz y Sandoval in Mexico first detected the parasitic nature of pinta. He believed there was only one species of fungus to be found in the affection and that the different colours of the patches were due to the different depths at which the fungus was growing in the various strata of the epidermis. Montoya's classical series and genera at each variety twenty different

species were found by him.

In the present state of our knowledge of pinta it is impossible to give a satisfactory classification of these fungi. The principal ones may be collected into the following groups —

- I Fungi of genus *Aspergillus* *Aspergillus pictor* Blanchard 1895 and several other species. *A. pictor* is found in the pure violet variety of pinta, the other species are observed in the pure blue and bluish and violet black varieties as well as in a form of the red variety. Several of these species are not in reality true *Aspergilli* as they possess organs intermediate between those of the genus
- II F . . . . . 1921; Castellani found in some greyish-violet varieties of pinta
- III Fungi of genus *Monilia* *Monilia montoyae* Castellani, 1907 Found in some cases of white pinta
- IV Fungi of genus *Montoyella* *Montoyella nigra* Castellani 1907 Found in one variety of black pinta. *M. boxini* Castellani, 1907 Found in a red variety of pinta.

PINT 4

term *Aspergillus* (*Trichophyton*) *peter* introduced by Blanchard in 1871 when the plurality of species of the fungi found in pinta had not yet been demonstrated is now used in a restricted sense to indicate the *Aspergillus* species from those found in Mexican pinta or carate.

**Appearance of the Fungi in Fresh Preparations**—Scrapings from Pitches examined in liquor potasse show in most cases between outer and thicker branches in comparatively large fructifications. The more typical characters of these fructifications vary according to the species and genus of the fungus present. They may be typical *Aspergillus* or *penicillium* like fructifications or they may show intermediate characters between those of *Aspergillus* and *Penicillium*. In many cases the fructification organ is represented by a pear shaped or triangular formation surmounted by five to six spores. The number of these spores however may vary. They are globular with a smooth surface showing a double contour and their diameter is much larger than that of the mycelial tubes. In the cases where the fungus present is a *Monilia* or a *Monodelia* such or similar fructifications are absent and only mycelial tubes and some scattered spores are seen.

**Cultures** The various fungi found in pinta are easily cultivated the best medium being Sabouraud's maltose agar. The optimum temperature is between 30 and 40 C.

The composition of Sabouraud's medium is —  
4 grammes  
1 gramme  
1.5 grammes  
tr. cc

Maltose  
1 cc Yone (Chang) no  
Agar  
Distilled water

- Culturally the fungi may be divided into three groups —
- 1 Those showing in cultures *Aspergillus* fructifications
  - 2 Those showing *penicillium* fructifications
  - 3 Those showing intermediate fructifications between the *Aspergillus* and the *Penicillium*
  - 4 Those showing simpler fructifications characteristic of the genus *Monilia*—viz a mycelial thread terminating in a single ring or small bunch of roundish spores.
  - 5 Those in which higher organs of reproduction are absent and the reproduction takes place somewhat similarly to what is observed in the genera *Uromyces* and *Trichophyton* by conidia and terminal segmented and unsegmented spindles. The fungi of this group are called by Montoya in a variety of black pinta by Robin and later by Cavellani in a variety of red pinta constitute the genus *Monilia*.
- In a series of experiments—Montoya has tried to infect callus and various cultures of the fungi found in the disease. On several



occasions = desquamation of skin and loss of hair was observed

temperature is constantly high. He states that he obtained pure cultures of the pinta fungi direct from such waters. He has also found the same fungi as ectoparasites on the bodies of mosquitoes of the genus *Culex* on sandflies (*Simulium*) and on the body of some bugs (*Clinocoris*) which are very common in the mines. He believes therefore that mosquitoes and other insects play a rôle in the transmission of the disease.

In some old chronic cases of pinta an *Acarus*—somewhat resembling *Acarus scabiei* though larger—has been found to live in the epidermal squame and some writers believe that this *Acarus* also plays a part in the transmission of the malady.

*Predisposing Causes*—What the older authors believed to be the true causes of pinta—viz a hot damp climate insanitary surroundings and poor feeding the mineral salts contained in the waters—are only predisposing causes some of which however are of great importance. The hot damp climate favours the growth of the fungi the water of the mines which contains a large amount of mineral salts (especially sulphates) produces after a time in those who use it for washing etc a dermatitis with fissures and other eczematous like lesions which greatly facilitate the infection.

All races are liable to be attacked by the disease but mulattoes seem to be particularly prone to become infected. Albinos are said

violet variety

S 1100 1691 11 T 1

11 11

ginous spots appear on uncovered parts of the body. The spots increase very slowly in size and some may fuse together. They are roundish or may have an irregular outline. At first they are hardly raised above the normal skin. The surface of the patches is generally dry and rough and is covered with fine pityriasis squamæ in recent cases with larger and thicker scales in older ones. Occasionally in chronic cases the surface of the patches instead of being dry may be moist or somewhat greasy or glutinous. The hairs of the affected regions become atrophied and later on

has been compared to the smell of cat's urine or to the bad odour of dirty linen kept in a warm damp place.

The affection may spread to the whole body except the palms of the hands and the soles of the feet. The nails are never attacked and the scalp is not usually affected.

The disease has no tendency to spontaneous cure. Its course is chronic and may last the whole of the patient's life.

Some of the older authors state that the patients during the incubation period suffer from fever, vomiting and diarrhoea. Montoya says that pinta patients have not got an odour *sus generis* as stated by most observers. In many patients no smell whatever is noticeable apart from the peculiar odour of the negro race.

**Local Varieties**—Clinically six different varieties may be distinguished, each of which shows several subvarieties—

- 1 The Black Variety
- 2 The Blue Variety
- 3 The Violet Variety
- 4 The Red Variety
- 5 The Yellow Variety
- 6 The White Variety

**Black Variety**—The patches are of a black colour and are very

common in the Asian race. The course is very chronic. The treatment is simple though not so difficult as in the other varieties.

Black pinta shows two subvarieties—one is characterized by the presence of patches of a black violet colour, the other by patches of jet black, Indian ink black colour. The fungus found in the black variety is an *Aspergillus* (species undetermined) in the record a *Morula* (*M. nigra*).

**Blue Variety**—This is much less frequent than black pinta. The patches are of a blue colour. They generally begin to appear first on the dorsum of the hands and then tend to spread over the whole body—uncovered as well as covered parts. There generally is intense pruritus.

The fungus usually found in blue pinta is *Aspergillus*.

**Violet Variety**—Apart from the colour of the patches which is violet the clinical symptoms and course of this variety are identical with those of blue pinta. It is extremely common among rural labourers and miners.

There are numerous subvarieties of violet pinta. In some cases the patches are of a pure violet colour in other cases the colour may be violet greyish violet brownish violet purplish. There are cases in which the patches are at first of a greenish colour to become violet bluish later on. The fungus found in the pure violet pinta is an *Aspergillus* (*A. pictor* Blanchard 1895) the fungus

**Red Variety**—This is the commonest variety found in white patients. The patches first develop as a rule on the dorsum of the hands and feet and spread to large portions of the body. The patches are red—often brick red—and usually show a rather abundant desquamation. Pruritus is very distressing especially at night time. Secondary lesions due to scratchings and inoculation of pyogenic micro organisms are not rare. Ulcerative lesions

Castellani in 1907 found of red pinta observed in a America. In this case the red patches there was on which a fungus was found (*P. montoyas*)

Red pinta is more serious than any other variety as it affects not only the superficial strata of the epidermis but the rete Malpighi as well as the corium.

**Yellow Variety**—Very common among half castes. It generally begins on the chest or arms. The patches are yellow and at first are not pruriginous and not desquamating. In old cases however there is pruritus. This variety is very frequently mixed with patches of white pinta and is difficult to cure. The fungi found

variety of pinta to represent in many cases the ultimate regressive stage of all the other varieties except the red. The patches of white pinta would be in such cases in reality unpigmented

leucoderma-like areas due to the disturbing action of the various fungi on the pigmentation processes of the skin.

Montoya's belief on the nature of white pinta in some cases is supported

*Mixed Variety*—Not infrequently the same patient may be affected with several varieties of pinta, presenting a grotesque tattooed or piebald appearance.

It is especially white pinta which is found associated with one or more of the other varieties.

*Diagnosis*.—This does not present any difficulty in the countries where the disease is endemic. In any doubtful case the micro-

The microscopical examination of scrapings from patches of *tinea nigra* will show mycelial tubes of irregular shape and large globular spores collected in bunches. The spores are grouped together in a somewhat similar manner to what one sees in *pitryiasis versicolor*.

*Tinea Flava*—In contrast to yellow pinta the fungus of *tinea flava* is a *Malassezia* has the same morphological characters of the fungus found in *pitryiasis versicolor* and cannot be grown artificially.

*Tinea Albigena and Tinea Alba*—These are generalized trichophytic and epidermophytic affections and are easily distinguished by the characters of the fungi.

*Leprosy* can be distinguished from white pinta lesions by the

situating them is often hyperpigmented.

*Prognosis*.—Pinta though not a fatal disease must be considered a serious affection as its course is chronic and the treatment very difficult. In most cases the general health remains satisfactory, but the disfigurement is very distressing to the patients, who often become nervous and irritable. The pruritus which is generally more marked at night is also a cause of great distress and sleeplessness.

have been of the nature of a trichosporosis (chrysoarobin = 10 parts)

tion The urine should be regularly analyzed during the treat

advisable, or citrine ointment may be used

Montova recommends chrysoarobin dissolved in chloroform (chrysoarobin 10 parts, chloroform 90 parts) Apply with a fine brush When dry, apply gutta percha dissolved in chloroform (gutta percha 10 parts chloroform 90 parts)

Chrysoarobin may conveniently be applied in the form of a verniceol varnish (5 to 10 per cent)

### PIEDRA.

**Synonym.**—Trichosporosis Tropica

**Definition.**—Piedra is a mycotic disease of some parts of South America causing very hard small nodosities on the hair

**Historical and Geographical.**—The condition has been known locally in Columbia since remote times, but the first scientific descriptions are due to Desenne (1878) Morris (1879), Osario and Megalhes More recently the condition has been studied by

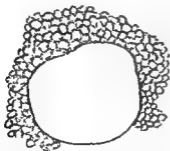


FIG 841.—TRANSVERSE SECTION THROUGH A PIEDRA NODULE



FIG 842.—PIEDRA

Juhel Renoy Pernet, J M H MacLeod, Horta and others This disease of the hair is common in some districts of Columbia especially the valley of Canca, but closely allied conditions are

observed in several parts of the tropics, and occasionally in

it is washed in ether, and then examined microscopically, the nodules will be seen to consist of large polyhedral refringent bodies held together by an amorphous substance acting as cement These bodies are the spores of the fungus causing the disease (*Trichosporum giganteum* Behrend, 1890) The description of the fungus is given on p 1101 In Columbia it is generally believed that the infection takes place by washing the hair with a mucilaginous oil, much used by the women of the country In British

Guiana natives consider it to be due to bathing in certain white or milky waters, while it does not occur if they bathe in the brown peat bush waters

matting and knotting of the hair The disease is chronic, and the nodosities do not disappear spontaneously

Horta has described a variety of piedra in Brazil, characterised by the nodules containing large cyst like structures, which Finoy considers to be probably asci (see p. 2102)

**Diagnosis.**—The microscopical examination of the nodosities renders the diagnosis easy

**Prognosis.**—The affection is of long duration and has no tendency to spontaneous cure

**Treatment.**—The treatment of this disease is not very satisfactory. It is best to remove the nodules by means of a pair of forceps, and to apply a solution of iodine to the raw surface. The hair will grow again, but it will be shorter and will be more brittle than before.

#### Trichosporosis India.

In India and Ceylon a condition similar to piedra is occasionally observed. It is, however, much less severe, a few minute nodules only being present on the hairs of the beard and moustache and the hair of the scalp being rarely affected. The fungus seems to be different from that of the Columbian piedra.

#### Trichosporosis of Temperate Zones.

Cases of trichosporosis have been reported from various parts of the world, including Europe, America, and Australia. The condition is characterised by the presence of small, white, nodules on the hair, which are composed of a mass of fungal hyphae. The nodules are usually found on the hair of the scalp, but they may also be found on the hair of the beard and moustache. The condition is usually of long duration and does not tend to spontaneous cure.

### TRICHOMYCOSIS FLAVA, RUBRA, NIGRA.

**Synonyms.**—Trichomycosis villaris, Trichonocardiasis, Tropical lepothrix, Castellani's Trichomycosis, Trichomycosis chromatica, Chromotrichomycosis.

**Definition.**—A nodular disease of the hair, characterised by the presence of small, white, nodules on the hair, which are composed of a mass of fungal hyphae. The nodules are usually found on the hair of the scalp, but they may also be found on the hair of the beard and moustache. The condition is usually of long duration and does not tend to spontaneous cure.

by European observers under various names, such as Lepothrix (E. Wilson), Trichomycosis nodosa (Patterson), Trichomycosis palmellina (Pick), but a great deal of confusion has existed until

recently on the subject very different clinical descriptions having been given and the condition being ascribed to widely different germs

Paxton Wilson Pick and later Payne Patterson Crocker Pusey etc described the hairs as presenting irregularly lobed masses of hard consistency in which were often embedded some of the fibres of the cortex

According to Crocker the fibres of the whole shaft may be split up and the hair may break off with a brush like termination The

micrococcus by Colombini etc Babes Pick Balzer and Barthely considered that the *Bacillus prodigiosus* played a rôle in the causation of the affection

In 1911 Castellani carried out an investigation in the tropics describing the condition as seen there and differentiating three varieties—the yellow variety the black variety the red variety He demonstrated that the yellow variety was caused by a nocardia (*Nocardia* or *Cohnistrepptolrix tenuis* Castellani) the black variety by the same nocardia plus a black pigment producing coccus

confirmed and amplified in the Sudan by Chalmers and O Farrell who suggested for the affection the term trichonocardiasis in West Africa by Macfie who described a variety of the red type *fusca* and by various observers in several other countries In 1915 1918 Castellani observed in the Balkanic Adriatic Zone the three varieties he had described in the tropics and found the same organisms

**Ætiology**—The researches of Castellani have demonstrated that the yellow variety is due to a very thin bacillary like fungus for which he proposed the name *Nocardia tenuis* later changed into

in the red type

# TRICHOMYCOSIS VILLARIS FLAVA

Characters of the Coccus like Organism found in the Black Variety (*Micrococcus* or *Nigrococcus nigrescens* Castellani 1911).—It is a Gram positive rather large non motile coccus, which in certain media may take the appearance of a coco-bacillus. Sugar media are more suitable for the growth of the organism than the ordinary agar.

- Sabouraud Agar**—Colonies appear twenty four to forty eight hours after inoculation. They are roundish at first white, but after a couple of days the centre of each colony turns black and thus pigmentation slowly spreads eccentrically. After a time the colonies coalesce into a jet black mass. The Glucose—Growth develops from the centre of the colonies and slowly spreads towards the periphery.
- Ordinary Laboratory Agar**—Growth much less abundant and does not spread to the whole of the growth.
- Leculose Agar**—Identical to glucose.
- Saccharine Agar**—The pigmentation is less pronounced and does not spread to the whole of the growth.
- Raffinose Agar**—Same as saccharine.
- Lactose Agar**—Black pigmentation well marked though in many cases it does not extend to the whole of the growth.
- Alkaline Maltose Agar**—Black pigmentation less pronounced than on acid maltose.
- Acid Maltose Agar**—Growth less abundant than on acid maltose. Black pigmentation well marked.
- Yeast Agar**—As alkaline maltose but pigmentation after a time becomes black.
- Inulin Agar**—As inulin.
- Saccharose**—As inulin.
- Glycerine Agar**—Abundant growth the whole of which after a time becomes of jet black colour.
- Galactose**—As inulin.
- Adonis**—Like acid maltose.
- Serum**—Growth fairly abundant but there is only a trace of pigmentation. The growth on the surface shows after the medium is not liquefied.
- Gelatine**—No liquefaction but the colonies on the stab are white.
- Milk**—No change.
- Broth**—General turbidity. A thin pellicle is often present. The microscopical examination shows cocci arranged in pairs or irregularly. They are not capsulated.
- Pepsine Water**—Some growth at the bottom while the rest of the tube clear.
- Sugar Broths**—No formation of acid or gas.
- Indol**—Most strains produce a trace of indol.

Characters of the Coccus like Organism found in the Red Variety of Infection.—The coccus observed in the black type of the infection and grow than the coccus observed in the black type of the infection a rule it grows better and shows more pigment on ordinary agar than sugar media. It is non motile and Gram positive.

**Agar**—The growth is at first white then a red or red yellowish spot appears in the centre. The pigmentation very slowly progresses to the periphery but seldom if ever spreads to the whole of the growth. Maltose and glucose agar the same pigmentation is present but on other sugar media no pigment is produced. Gelatine and serum liquefied. This coccus as already stated is Gram positive and non motile. The coccus has been recently further investigated by Chalmers and who have observed that the best medium for showing the pigment is if they have called the coccus *Red coccus castellani*.

**Symptomatology.**—The affected hairs of the axilla are firmly plucked to the naked eye of the patient. They are easily removed by scraping with a scalpel.



disease was commonly known in some parts of India as 'Madura foot'. As no mention is made, as far as we know, by these authors of any black pigment being present in their cases, we conclude that probably they saw the actinomycotic variety of mycetoma

it this time (1845) von Langen

some curious bodies which he

and which he found in the pus

from a case of spinal caries. Unfortunately he never published this observation, which was made known by Israel one year after

Bollinger

In 18

about th

Louis h.

of the t

bodies were carefully examined both microscopically and chemically, and drawings were made which were subsequently published by Lebert (1857)

We have examined copies of these drawings and they represent in a typical manner the fungus of an actinomycosis. Lebert, however, failed to recognize their fungal nature.

In 1855 Smith, in London, made some drawings for Paget of a tumour of the upper jaw, in which an organism resembling a ray fungus is portrayed. These drawings were published by Kanthack (1896)

Also in 1855, Ballingall in India, described a disease of the foot in the discharge from which he found bodies composed of large cells with transparent fringes containing irregular spicules or simply composed of radiating spicules without cells. In 1858

substance Rustomji's first variety we call *yellow actinomycosis* and his second variety *black maduromycosis*

We will now consider the subject of the Maduromycoses

### THE MADUROMYCOSES.

**Definition.**—The Maduromycoses are those forms of mycetoma

sidered when we discuss that author's writings, the history of the

looked upon it as a local tubercular affection, and, influenced by this view, he considered the black particles mentioned above to be accidental, and not essential parts of the disease. He also mentions that it was known to the natives as 'ghootloo mahdee,' from the tubercular irregularities being supposed to resemble eggs.

This first case of black maduromycosis occurred in a native aged

Carter says that the second volume of the 'Indian Annals' (probably dated about 1849) on p 706 contains an account of

many years.

It may perhaps be advisable at this point to draw attention to the fact that Haltingall's celebrated observations do not refer to the black, but to the yellow variety of mycetoma, and hence do not enter into this history.

Sub-Assistant Surgeon Bazonji Rustomji (1858), of the Bhoo's Dispensary, in the Province of Kutch, drew attention to the fact that there were two forms of the disease—viz., one in which there was no granular deposit, but only a substance dark in colour and

In 1860, Vandyke Carter began a series of classical observations upon the black and yellow forms of Madura foot, which he continued until 1874, and during which he firmly established the fungal

..  
 differentiated between the white or ochroid division of the Mycetomas, which to day we call 'actinomycosis,' and the black or melanoid variety, which we now name 'black maduromycosis'. He demonstrated that the black grains were of true vegetal nature with a black friable rind composed of clear, orange-tinted, ovoid

..  
 sclerotes was composed of slender, pale, flattened, and branching fibres arranged in bundles and intermixed with numerous granules and a few large beaded fibres, the septa of which were sometimes absent.

..  
 clear connection with the fungus particles, but seemed to spring up independently of them upon the rice whenever this was exposed to the air'

formibus.'

..  
 The ... of ... was never recognized by myco-  
 Mortierella  
 i contami-  
 was never

..  
 first proof  
 of the parasitic nature of the grains he was unable to produce growths by cultivation from either the black or the white varieties

'dégénération endémique des os du pied'

H. J. Carter (1862) came to the conclusion that the fungus of

had been embedded for two years. In the same year, Bristowe described and figured the fungus seen in the black particles of a foot from a case of black maduromycosis amputated in Canton, and demonstrated to the Pathological Society of London by Tilbury Fox. Bristowe's descriptions and figures are excellent, and amply confirm Vandyke Carter's work. Thudichum chemically examined the black pigment of this case, and showed that it was not derived from blood.

Hogg (1872) described a black maduromycosis from India, in which he was able to observe the fungal threads and to resolve them into jointed dissepimented cells, some branching out and attaining a considerable length, while others terminated in an enlarged or old head. He, however, believed that the fungus was a secondary

the black particles, but not of the yellow granules. They showed that *Chionophloeocarteri* had nothing to do with black or yellow grains.

In 1876, Berkeley came to the conclusion that *Chionophloeocarteri* had nothing to do with mycetozoa, a point which can be easily judged from the passages quoted above.

great deal of confusion  
 which can be judged by  
 It was admitted that

particles, and because there was not sufficient evidence forthcoming at the time in proof of the vegetal character of the yellow grains which were believed to be essentially fatty in nature. It was, however, admitted that Moore's observation showing that the black variety could be cured by excision of all the particles at an early stage of the disease was a strong argument in favour of the parasitic nature of mycetoma.

Though Carter had found black, yellow or white, and red grains, still the general belief was that these were one and the same process and, moreover, observers of this period must have seen the pseudo-mycetomatous conditions mentioned above, because competent workers appear to have met with cases in which they were unable to find any grains, although the clinical appearances resembled mycetoma.

Corre (1883) placed in order, completed and revised the notes of researches made by Collas since his publication, already mentioned, in 1861. In these notes which were published after his death, Collas desired his previous name for the disorder to be altered to 'La Maladie de Balingall,' and states that the earliest references to the disease with which he is acquainted can be found in Waring's paper, and in one of the sacred books of the East which he calls 'Vaweda' (Ushta wunga hrethayum) which appears to us to be the 'Atharvaveda.' In this latter work sliptham or elephant foot is distinguished from 'padavalnicum,' which refers to an incurable malady of the foot associated with swelling and the formation of

'leg of an elephant' In Bandy, he says, the word used was 'gootloo mahdee,' because the swellings on the foot were thought to be like eggs, while in Rajputana it was called 'kirmagra,' or the dwelling house of worms, because the sinuses were considered to be like the cavities often occupied by the larvæ of flies. He also says that in 1714 a missionary described under the name of 'fourmilere des vers' a disease of Pondichéry which was incurable and in which numerous ulcers intercommunicated by means of small canals full of worms, which were peculiar in that if one closed  
 red from vol II,  
 titled 'Memoires  
 sionaire.' Collas

### Ballingall's disease

With reference to the above names, it will be noted that they apply to any form of mycetoma and not especially to black mycetoma. The name 'Ballingall's disease' in our opinion, is not applicable to the black mycetomas because as already indicated he was not acquainted with the disease.

In 1886 Carter gave up his pink mould and drew attention to the similarity between the fungus of actinomycosis and that of mycetoma.

consist of an olive brown glassy or finely granular material, in which hollow filaments radially arranged were embedded, still he regarded these as degeneration changes and sought to prove

fungus of the yellow variety existed in the black, while the former observer believed the reverse to be true. He named the fungus *Oospora indica* Kanthack 1893 and distinguished the two varieties as *O. indica* var. *flava* and *O. indica* var. *nigra*. Unna to whom he sent specimens however did not make this error, but says —

A whole series of important distinctions separate the two fungi, and there is no question of their identity.

Hoyce and Surveyor (1891) in a most important paper first definitely proved that the fungi existing in the black and yellow varieties were quite different, and thus definitely established the two main divisions of mycetoma which to-day we call maduro mycosis and actinomycosis. They showed that the black grains were composed of a large septate branching fungus embedded in a brown pigmented ground substance which was readily bleached by eau de Javelle. They did not observe spore formation nor was cultivation attempted.

In the same year Boccro also differentiated between the white

broth, small white colonies composed of radiating threads were found sticking to the walls of the tube. No diffuse growth was seen, nor did any scum form on the surface. Animal experiments were negative.

Mackenzie, in the same year, appeared to obtain similar cultures on agar, at first the growth was white and translucent, with radiations from the centre, later it became greyish yellow, there being a central granule surrounded by a clear zone and an indented margin. After a week the colony became a deep mahogany, and under the microscope exhibited mycelial structures.

Semon (1915) reported a case of black maduromycosis which occurred in a native Indian soldier serving in France. He left India about October, 1914, and in January, 1915, he injured one of his feet by the fall of an ammunition box. The patient attributed the disease to this cause, but Semon considers, probably correctly, that he must have been infected before leaving India. A typical mycetoma developed in about six months, and the pus contained

marked vascular hypertrophy, polymorphonuclear, plasma, and connective tissue cells but no endo- or periarteritis and no giant cells. Growths were obtained at 35° C on agar agar, maltose agar and Raulin's fluid. The fungus formed a central black portion with a peripheral zone of white or grey and in the course of ten days or less became black.

In 1916 Chalmers and Archibald grew a fungus allied to that described by Semon from a case of black maduromycosis found in the Anglo-Egyptian Sudan and in 1918 defined and classified the Maduromycoses.

**Climatology.**—The Maduromycoses are known to occur in Europe, Africa, Asia, and America, but not in Oceania.

The climatology has been most thoroughly studied for black

Balfour's researches in 1911 have already been noted in the historical

list of African places from which cases of black maduromycosis have been reported—Algeria, Tunisia, Somaliland, Madagascar, Transkei (South Africa), Senegal, and the French Sudan.

In Asia the disease is recorded from the Yemen, various parts of India, Ceylon, and possibly from North Borneo.

In America it has been described in the United States by Wright, and in the West Indies by Scheult

In Europe it has so far only been found in Italy, Macedonia, and

into six zones of vegetation, viz —The Northern Glacial Zone, the Northern Cold Winter Zone, the Northern Hot Summer Zone, the Tropical Zone, the Southern Hot Summer Zone, and the Southern Cold Zone

contains wet areas. The black maduromycoses are most commonly

Africa is mostly moist

floral regions viz —India, Persia, India, Persia, India, Persia

Dissects the present forms of the

the climate is hot and arid

The Southern Hot Summer Zone includes South Africa where the disease has been recorded, but where it is apparently rare

This is as far as the present state of our knowledge permits us to go with regard to geographical distribution, and more research



broth, small white colonies composed of radiating threads were found sticking to the walls of the tube. No diffuse growth was seen, nor did any form on the surface. An animal experiment was negative.

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marked vascular hypertrophy, polymorphonuclear, plasma, and connective tissue cells but no endo- or periarteritis and no giant cells. Growths were obtained at 35° C on agar agar, maltose agar, and Raulin's fluid. The fungus formed a central black portion with a peripheral zone of white or grey and in the course of ten days or less became black.

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Cold Zone

contains wet areas. The black maduromycoses are most commonly

floral regions, viz —India Deserta, India Diluvra, India Aquosa, India Vera, India Sub-Aquosa, and India Littorea, while black

Region consisting of the dry but not desert triangle between the Western and Eastern Ghats with its apex at Tinnevely and its base at the borders of the plain of the Ganges.

The white varieties of mycetoma are also found in this area, but are outnumbered by the black maduromycoses while in India

the climate is hot and arid

The Southern Hot Summer Zone includes South Africa, where the disease has been recorded, but where it is apparently rare

This is as far as the present state of our knowledge permits us to go with regard to geographical distribution, and more research

on this part of the subject is required, but from the above it is obvious that heat and aridity are favourable conditions for the fungus

of microscopic specimens

the presence of coloured

reddish or yellowish in

*maduromycosis*

These coloured granules are called 'grains,' a term which has been defined by Chalmers and Archibald as follows —

'The term "granum" or grain has been given to differently coloured bodies of varying consistence, size, and shape, found in mycetomas and composed of hyphæ with sometimes chlamydo spores, embedded in a matrix and giving rise to mycelial filaments on germination.'

The ætiological importance of these grains and their contained fungus rests upon the fact that they are present in all forms of

grains

III The *red maduromycoses* with red grains

### I THE BLACK MADUROMYCOSES

These may be divided into —

- A The *European black maduromycoses*
- B The *African black maduromycoses*
- C The *Asian black maduromycosis*
- D The *American black maduromycoses*

#### A THE EUROPEAN BLACK MADUROMYCOSES

(1) *Bassini's Kobner's* and *Schmincke's black maduromycoses*, respectively found in Padua, Italy, and in Kissingen, and of which the nature of the ætiological fungus is unknown

(2) *Boro's black maduromycosis*, found in Genoa, and of which the causal agent is called *Madurella boro* Brumpt, 1910, but this

is not the same as the one described by

(3) *Peperé's black maduromycosis*, found at Domusnovas in the Province of Cagliari in Sardinia, and caused by *Scedosporium sclerotiale* Peperé, 1914

### B THE AFRICAN BLACK MADUROMYCOSIS

(1) *Brumpt's black maduromycosis*, caused by *Madurella mycetozoa* (Laveran, 1902)

(4) *Chalmers and Archibald's black maduromycosis* caused by *Glenospora khartoumensis* Chalmers and Archibald, 1916, which has now been recovered three times in the Anglo-Egyptian Sudan

These African black maduromycoses may be differentiated from one another as follows —

A. Microscopical preparations show aspergillar heads—*Bonford's black maduromycosis*

B. Microscopical preparations do not show aspergillar heads, on culture the following types of spore are obtained —

1. The alicornosporal form of conidium—*Chalmers and Archibald's black maduromycosis*

2. The arthrosporal form of thallospore—

(a) Mycelium greyish white when old yellowish and darkening

in the cultures Up to the present the inoculation into animals is negative Very widely spread in Africa

(f)

## C. THE ASIAN BLACK MADUROMYCOSIS

There is only one type known at present—viz, *Carter's black maduromycosis*, caused by *Glenospora semoni*: Chalmers and Archibald 1917, which can be readily differentiated from *Glenospora khartoumensis* Chalmers and Archibald, 1916 by the following characters:

- (1) Grown on clear maltose agar in Khartoum after twelve days in an

with hardly any white fringe

- (2) and (3) Grown on glucose agar and blood serum, there are marked differences between the two fungi

## D THE AMERICAN BLACK MADUROMYCOSIS

(1) *Wright's black maduromycosis*, which was found in the United States in an Italian woman who had left Italy, where black maduro-

mycosis is a native of India who had left that country twelve years before the onset of the malady. The nature of the causal organism is unknown.

## II THE WHITE OR YELLOW MADUROMYCOSIS

These may be divided into —

- A The *European white maduromycosis*  
 B The *African white maduromycosis*  
 C The *Asian white maduromycosis*

## A THE EUROPEAN WHITE MADUROMYCOSIS

(1) *Brumpt and Reynier's white maduromycosis*, caused by *Indiomyces reynieri*: Brumpt, 1906 with a large soft grain found in Paris

(2) *Tarozzi and Radaeli's white maduromycosis*, caused by *Scedosporium apiospermum* (Saccardo 1911), with a small rather hard and yellowish grain, found in Sardinia and Italy

## B THE AFRICAN WHITE MADUROMYCOSIS

*Nicolle and Pinoy's white maduromycosis*, due to *Sterigmatocystis nidulans* (Cidam, 1883) with grains of size varying from those which are almost microscopic to others about the size of a pea in rounded or polyhedral form, and of variable colour, being dirty white or yellowish white, and soft in consistence, and found in Tunisia





## C THE ASIAN WHITE MADUROMYCOSIS

Brumpt's white maduromycosis, due to *Indiella mansoni* Brumpt 1905, with very small and very hard white grains found in India

The differentiation of the white maduromycoses may be effected as follows:—

## A Grains soft —

- 1 Sterigmatocystic heads found in grains and in cultures. Grains not like a ribbon rolled on itself—*Nicola's* and *Pinkoy's* white maduromycosis
- 2 No such heads to be found in the grains which are like a ribbon rolled upon itself—*Brumpt's* and *Reynier's* white maduromycosis

## B Grains hard —



## III THE RED MADUROMYCOSIS

Only one form is known *Balfour and Archibald's red maduromycosis* which was possibly due to an aspergillus because aspergillar like heads were found in the grains. It occurred in the Anglo Egyptian Sudan.

**Pathology**—The causal fungus is introduced into some part of the body by a wound produced by a thorn, a splinter of bamboo or other wood by a sharp stone knife etc. but once introduced into the subcutaneous tissues it commences to grow the original wound in the meantime healing. Usually the growth is slow, but if

purative lesions. As the fungus grows it destroys the tissues of the foot and meets with but little reaction on the part of the body,



later, breaking free from the main mass, form means of asexually  
 clubs  
 ssible  
 rches

show that the leucocyte, with its enclosed club, may wander away from the diseased area into healthy tissue, and may be killed by the club, which, being set free, grows into a new mass of fungus. In this way the fungus may be disseminated by means of the phagocytes.

When surrounded by pus, the fungus gathers itself into granules the so-called sclerotia, which show externally radially arranged

form of degeneration and disappears. The result is that the foot appears much swollen externally, and shows the openings of the sinuses through which the pus and the fungoid granules escape, while internally the normal structure may have completely disappeared, being replaced by degenerated tissue, debris, sinuses and fibrous sacs containing the fungus and the pus.

The result is however, not merely destruction of the foot, but also great bodily waste, due to the continuous discharge, so that the patient becomes emaciated, and may finally die of cachexia.

**Morbid Anatomy.**—The pathological anatomy of black maduromycosis has been the subject of a fair amount of investigation. Kanthack merely drew attention to the fact that the black masses were always to be found embedded in dense fibrous tissue, while a few pus and granulation cells were to be seen in most cases. In the fibrous wall yellowish brown or black pigment could be found while fuchsin bodies were present in most specimens. Unna's example, obtained from Kanthack, only showed fibrous and some granulation tissue. Boyce and Surveyor drew attention to the presence of small round cells macrocytes and giant cells surrounding the fungus in cases of black maduromycosis. Their microphotographs are, however, mainly devoted to the fungus while their

more or less  
 granules lay  
 epitheloid cells,  
 or a wall of  
 vascular granulation tissue or by masses of epitheloid and multi-  
 colored or other granules,  
 he gives  
 which

Figs 4, 5, and 6, though older, if examined with a lens, will be seen to agree more or less with Boyce and Surveyor's Fig 22

Oppenheim's description in 1904 mainly deals with the fungus, but Brumpt's account of the histological changes induced by *Aspergillus bouffardi* covers all the important points—viz, the polymorphonuclear leucocytes, the lymphocytes, the giant and

cells are situate close to the fungus

Boccardo, writing in 1909 in general terms for the encapsulated form of both white and black mycetomas, says—

This description, which unfortunately is not illustrated, agrees

formed by the falling out of some of the black granules during preparation. The spaces demonstrate the character of the lacunæ occupied by the grams and their

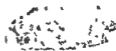


FIG 845  
BLACK MADURO MYCOSES

some  
fact  
uppe  
also  
fungi

cells, bloodvessels, and lymph spaces, the last mentioned being being markedly partially shown, continuous with



FIG 846.—BLACK MADUROMYCOSIS

1. Fungus
- 2 Small cells
- 3 Giant cells and large cells
- 4 Small cells, connective tissue, bloodvessels, and lymph spaces
- 5 Dense connective tissue

The cells are separated by a variable amount of fine connective tissue, which also supports large lymph spaces and bloodvessels. Débris and pigmentary granules can also be seen. A special rare feature of this layer is the presence of mononuclear cells containing one or more eosinophile rounded bodies, which were first observed in this pathological condition by Kanthack, and subsequently by nearly all the other workers on the morbid histology of the black

maduromycoses, to which they are, however, not confined. Their exact nature is unknown, but they are probably in some way due to the fungus.

On inspecting the upper part of the cellular mass, it will be observed that the white fibrous tissue increases in amount, but is still loose and contains many cells in its meshes, while more

latter at times show signs of endarteritis or periarteritis, by which means the lumen of the vessel may be considerably diminished or even closed.

Very rarely do the fungi invade the body, and rise to a general infection.

The peculiarity of the pathology is the slight reaction which the body makes against the invasion by the fungus and the entire absence of any attempt at repair.

The black varieties of mycetoma owe their colour to a dark

ALL THESE MYCEMATA UNDER THE SKIN USUALLY FORM NODULES AND

fibrous tissue formation, endarteritis, and periarteritis, and at times absorption of the bone.

THE M. PROCELOSPORA FORMS NOT A

numerous plasma cells and occasionally giant cells there is a marked proliferation of bloodvessels

**Symptomatology**—The disease usually begins in the foot more rarely in the hand and still more rarely in the leg knee neck or trunk There may or may not be a history of a cut or injury some time previously In any case this primary injury will have healed long before the disease is well established

The incubation period in well recorded cases would appear to be short thus in Musgrave and Clegg's case one month after the

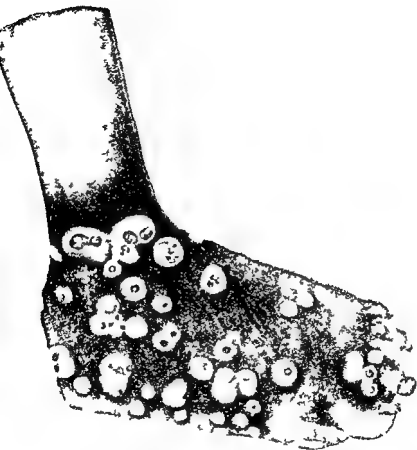


FIG 847—MYCETOMA

primary injury the wound reopened and discharged pus and in their experiments on monkeys it appeared to vary from ten to sixteen days Further researches on this point are however, required

... the region of the  
 ... in the  
 ... in the  
 ... small  
 ... is in

which the granules characteristic of the fungus may be found



MYCETOMA



Some new indurations and nodosities appear in various parts of the foot and new openings are formed while the whole foot begins to swell in a very characteristic manner. First the arch of the

nodules and openings. The colour of the integument may be

As the disease progresses pain which at first was slight becomes more marked especially in cold weather. The patient ceases to

The disease is very chronic and has no tendency to heal and if not treated will eventually cause the death of the victim from

to be diagnosed with certainty from elephantiasis or tubercular disease of the foot

not recur

If however the growth has lasted some time and has involved the bones the prognosis is not good and is worse if the lymphatic glands are also implicated

age and as com  
well above the  
of any enlarged



Potassium iodide may be tried but as a rule internal medicines and vaccines are useless for this form of mycetoma

**Prophylaxis.**—The wearing of boots and shoes and not walking barefoot are apparently good and sensible methods of prophylaxis.

### THE ACTINOMYCOSES.

**Definition.**—The Actinomycoses are those forms of mycetoma with grains composed of very fine non segmented mycelial filaments in which usually the walls are not clearly defined from the contents and in which chlamydospores are absent

**History.**—This period opens with Bollinger's epoch making work in 1876 on the lumpy jaw of cattle, a disease which had been recognized since 1785 as

of a branching organism

(1877-78), who gave it

fortunately, this generic name cannot stand, because, unbeknown to Harz it had already been used by Meyen (1827) for a fungus which he called *Actinomyces horkelii*, which is in no way related to the group of fungi which we are considering. This mistake launched the generic name applicable to these organisms on to a sea of change, and led to much confusion

1 *Nocardia bovis*—The correct name for Bollinger's organism is

*Nocardia bovis*

seen in man

attention to

variety of my

to demonstrate the presence of actinomycosis in man, and as Israel's name is associated with

propose to name this

Vandyke Carter, as we

to the likeness between

in 1891, Bostroem grew

in 1891, Bostroem grew

It grows well aerobically at 22° C., but better at 37° C. Anaerobic growths are, as a rule, but poorly developed

It may form a dry pellicle on the surface of broth, but more

resulting fluid may or may not be dark coloured. On blood serum it produces poor growths, and no liquefaction or pigmentation of the medium

On agar and glycerine agar it forms hard, spherical white colonies which give rise to an undulating crateriform growth having a yellowish or greyish tint which in its turn becomes a lichenoid ashen grey or yellowish mass with a powdery efflorescence. On maltose agar it forms discrete fawn coloured colonies later becoming yellow dark brown or even black while the medium may be slightly darkened.

On potato it forms confluent hard raised, variously coloured

and it is also experimentally shown that guinea pigs have been infected by intraperitoneal inoculation.

*Nocardia asteroides*—No and a lot of other species are also

*N. madure* (*N. indica*) but he does not appear to have done so

*uliginosa*

Also Cranwell, Bachmann and Del Pont (1909) gave an excellent and well illustrated description of a yellow mycetoma in Buenos

Aires Unfortunately, they did not grow it on inspissated blood serum but as far as we understand their account we should classify this organism which they did not name, as *Nocardia asteroides*

Nature of Test	<i>N. asteroides</i> from Musgrave and Clegg	<i>N. brasiliensis</i> Lundberg	Result of Comparison
Seat of disease	Mycetoma of foot	Mycetoma of leg	Difference unimportant
Grains	Consistency dough like colour yellowish lowish white size 0.25-0.5 mm in diameter	Consistency soft colour yellowish white size 0.1-0.3 mm in diameter	No important difference
Clubs	Usually absent	Absent	Agree
Bacillary and coccid forms	Numerous bacillary and coccus like varieties	Bacillary and coccid forms present	Agree
Optimum temperature	Slower growth at 30° C than at 37° C	Better growth at room temperature than at 37° C	Slight disagreement
Anaerobic cultivation	Does not grow	Does not grow	Agree
Broth	Floating flat particles which later fall to the bottom. Medium not affected	Small particles which later fall to the bottom of the tube. Medium not affected	Agree
Gelatine	No liquefaction	No liquefaction	Agree
Sabouraud's glucose agar at 37° C	Centre yellow periphery pink to pinkish white	Colonies rose violet	Slight disagreement
Potato			Agree
		periphery the medium becomes darkened	
		brown	
Serum	Growth slower. Colonies at white later pink	Grows very badly at 37° C as white	Later pink not mentioned in <i>N. brasiliensis</i>
Milk	Howish mass agglutination	with orange agglutination	Agree

## THE ACTINOMYCOSES

2135

*Nocardia asteroides* possesses Gram positive acid but not alcohol fast hyphae which are without club like enlargements. It produces restricted growths aerobically and usually anaerobically at 22° C and 37° C but nothing is stated in the literature we have consulted with regard to any odour arising from these cultures. It does not liquefy gelatine or blood serum nor has it any diastatic action. It reddens litmus milk which later becomes alkaline but is not coagulated or cleared. It grows on the agars and on potato producing reddish (often brick red) growths. It is pathogenic for monkeys rabbits and guinea pigs.

3 *Nocardia liquefaciens* — This fungus was obtained by Hesse in 1892 from a man in Germany with a left inguinal abscess which communicated with the rectum. Subsequently other abscesses formed on either side of the dorsal spine. The pus from these abscesses discharged soft yellowish grains about the size of a millet seed which contained a Gram positive fungus which did not possess clubs. On cultivation it grew readily and was found to be strictly aerobic. In gelatine stabs it formed a nail shaped growth which at room temperature in Europe was only visible on the third day while liquefaction beginning on the fourth or fifth day was complete by the end of the week. The liquefied gelatine was not discoloured and if the growth stuck to the glass it was yellowish with a whitish covering. On blood serum it formed small cloudy granules of the same colour as the medium in twenty four to forty eight hours. Liquefaction begins at the end of the first week and proceeds slowly the liquid remaining quite clear and colourless and only after some six months turning to a reddish yellow colour. In broth it forms delicate flakes which fall to the bottom of the tube and consist of a lower surface which is yellowish white and an upper surface which is snow white. The medium remains quite clear. No surface growth is mentioned.

On agar the colonies at first form separate rosettes which remain distinct for a time. These colonies appear to resemble the gelatine culture being yellowish below and having a white envelope. The growth on glycerine agar is more vigorous than on ordinary agar.

On potato it forms small yellow nodules by the second day which later become covered with a snow white efflorescence which does not alter. Apparently it was not grown on glucose agar media or eggs. Intravenous intraperitoneal and subcutaneous injections into rabbits guinea pigs and white mice were negative.

Hesse gave it the name *Cladothrix liquefaciens* which now becomes the organism as that named *Streptothrix buccalis* by Goadby in 1909 and found by him in 1899 in the mouth in cases of pyorrhea. Goadby's form showed clubs or club like swellings. It precipitates the casein in milk which became clear.

4 *Nocardia indica* — Kanthack in 1893 studying specimens of a new mycetozoa which came from India concluded that

Clegg and Hobby (1916) described *N. indica* in a native woman in Hawaii

*Nocardia indica*, with yellow or red grains, possesses Gram-positive but not acid-fast hyphæ, without clubs. It forms restricted growths under aerobic surroundings at 22° C. and 37° C., but will not grow under strict anaerobic conditions. The cultures are without any distinct odour. It is usually said not to liquefy gelatine or blood serum, but Koch and Stutzer say that it has a peptonizing effect after a long time. Milk is not coagulated, but after some time is cleared. Pinkish colonies are produced on the agars and on potato. It is non-pathogenic for animals, as far as is known.

5 *N. garteni*—Garten, 1895, met with an organism in cases of actinomycosis in man which he called *Cladothrix liquefaciens* No. 2 in order to distinguish it from Hesse's fungus, which he called *Cladothrix liquefaciens* No. 1, but Brumpt, in 1910, altered Garten's name to *Discomyces garteni*, which now becomes *Nocardia garteni* Brumpt (1910).

This fungus was grown in 1895 by Garten from the lesions of a case of necrosis of vertebræ and ribs, which was associated with abscesses, sinus formation, and empyema. The grains were com-

Nothing is said as to the liquid being coloured in any way, and, therefore, we must assume that it was not tinted. On agar, glycerine, and glucose agar it formed a greyish-white growth which became somewhat wrinkled on the surface after two to three days. The wrinkles are deep folds on glycerine agar.

On serum it forms a white layer, which becomes wrinkled and folded after forty-eight hours, when commencing liquefaction may be noted. On the third day the liquid has increased considerably, and by the sixth day the whole serum is reduced to a perfectly clear fluid. On potato it gives rise to white colonies, while the surrounding medium becomes greenish in colour. It apparently was not

cess  
ized  
the

diphtheria bacillus

It did not grow at 22° C. nor on gelatine or potato, but it was grown on colonies on mice (Chester, 1901). which name has become changed to *Nocardia krausei* (Chester, 1901).

Allied to, or identical with, this species are the fungi causing the conditions described by Mosevig-Moorhof, Dor, and Poncet, and often called 'pseudo actinomycosis' or the mycoses with yellow

grains which are larger than those of the ordinary actinomycosis while they are less numerous in the pus. Microscopically they show a tangle of filaments longer and larger than those of ordinary nocardias between which lie micrococcal like debris. They never show clubs at the periphery and do not grow on solid media like gelatine. They grow quickly in broth forming a skin on the surface. Cultures on serum give clavate forms like the diphtheria bacilli.

The fungus causing the above conditions was named *Nocardia ponceti* by Verdun in 1913 and may be a synonym for *N. krausei* (Chester 1901) for the following reasons —

A The pseudomycetomatous condition of Poncet does not differ from the definition of actinomycosis given at the commencement of this paper

B *N. ponceti* only differs from *N. krausei* in the following details —

1 Broth is rendered turbid and has a bad odour but Foulerton has pointed out that this turbidity together with the odour which was described as

absent

2 According to Verdun it does not grow on agar. It is not known whether *N. krausei* grows on plain agar but it can grow on glycerine agar and (according to some authors) on glucose agar

C They resemble each other in —

1 Morphology

2 They both possess clavate forms like the diphtheria bacilli

3 Both grow on serum

4 Neither grows on gelatine

macroscopical appearances of some of the ochroid varieties of

mycetoma, this variety might be found to be more common than Vincent's *N. madura* (= *N. indica* of Kanthack)

Balfour (1911) reported the presence of the same causal agent in a case of mycetoma of the foot in the Province of Swaziland and gave a photomicrograph of the same year Fulleborn

described a case from South West Africa, which occurred in a Herero aged twenty years. A study of Fulleborn's preparation induced Brumpt to alter his generic diagnosis for the fungus which, in 1913, he classified as *Discomyces somaliensis*, which, converted into our present nomenclature, becomes *Nocardia somaliensis* (Brumpt, 1906), but he is inclined to think that it ought to form a separate genus or subgenus, for which he proposes the name *Indiellopsis* Brumpt, 1913, because it secretes around itself in the grain a hard sheath, insoluble in potash and in eau de Javelle, which no other nocardia is known to do.

In 1916 we met with this fungus in a mycetoma of the foot in Khartoum.

The grains are hard, 1 millimetre in diameter, and being of a

beautifully illustrated account of a streptothrix, which they had isolated from two cases of actinomycosis in man—viz, from the organism was cultured anaerobically, that into animals were induced Kruse, in 1896, to make a new species for it under the name *Streptothrix israeli*. In 1911, for reasons presently to be set forth, Pinoy founded a new genus, *Cohnistreptothrix*, with Israel's organism as the type species, and therefore its name becomes *Cohnistreptothrix israeli* (Kruse, 1896).

## THE ACTINOMYCOSES

is to be found in all works of any importance on systemic mycology. Therefore as *streptothrix* is not available after many changes the generic name has become *Cohnistreptothrix Finoy* 1911 and to this genus Israel's human organism belongs. It suffers from Bollinger's type of fungus in growing best anaerobically in being difficult to cultivate and in not producing arthrospores. Other allied organisms are *Cohnistreptothrix thibergei* (Ravaut and Finoy 1909) also found in actinomycosis in man. *Streptothrix spitis* Lignières 1903 found in cattle and probably identical with *C. israeli* as may be Doyen's *streptothrix* while *Nocardia cerrogensis* Cougerot 1909 in juxta-articular nodules and *Streptothrix cuneatus* Schmorl 1891 probably also belong to this genus as well as the *streptothrix* recently discovered in a liver abscess in America by Bloomfield and Bayne Jones (1913) as we have consulted the authors upon this point with which they are in agreement. Perhaps the bacillus described by Sawtschenko in 1896 as the causal agent of a pseudo-mycetomatous condition may also belong to this genus and it is also possible that the *Caetobasilus pseudo-actinomycosis polymorphus* Berestneff 1895 may be the same as the chromogenic anaerobic *streptothrix* obtained from human pus by Neschewadzenko in 1908 and carefully described.

8 *Cohnistreptothrix israeli*.—This organism appears to be of increasing importance in human pathology for according to Finoy it appears to affect man more often than *Nocardia bovis*. It was first discovered in man as mentioned above by Wolff and Israel in Germany and has since been found in thirteen cases in the United States by Wright. It has also been found in cattle by Lignières and Spitz (1904) in the Argentine and by Finoy (1913) in France.

It is composed of short and long rods some of which show club like swellings while in old cultures spores which resemble cocci in appearance can be seen. It grows but poorly in the presence of air but much better anaerobically at 37° C on agar on which it forms dew like drops which later become yellowish and generally remain discrete. In broth it forms a deposit of small scaly particles. It does not grow on gelatine at the room temperature of Europe but egg cultures show typical branched filaments with club like ends which later break up into bacillary and coccid forms but no arthrospores (i.e. resistant spores) are not produced. It forms granulation tumours when inoculated intraperitoneally into rabbits and guinea pigs after an interval of four to seven weeks. In all tumours typical actinomycotic grains can be found containing branched filaments with clavate ends.

9 *Cohnistreptothrix thibergei*.—This fungus was discovered in 1909 by Ravaut and Finoy in a case of actinomycosis produced generalized subcutaneous and intramuscular nodules in a man in France.

The nodules opened and discharged blood tinged pus in which the fungus was seen sometimes in isolated bacillary form at times as very small white grains which in the tissues might be some 80 microns and be composed of a radiating mycelium without fine club forms. It grows well aerobically and locally but the former produces more bacillary and the latter filamentous forms. The optimum temperature is about 37° C. It does not appear to be pathogenic for laboratory



**Climatology.**—The geographical distribution of this variety of mycetoma is as follows —

I *Black actinomycosis* Only one variety of this is known—  
viz —

*Babès and Mironescu's black actinomycosis*, found in Roumania and caused by an unknown fungus

II *Yellow actinomycosis* This form is well known in North America and Sutton, of Kansas City, in 1913, in addition to drawing attention to four previously described cases, added two from his own practice. The usual microscopical

below are the nine varieties known to us are —

1 *Israel's yellow actinomycosis* found in Europe and America (North and South), and caused by *Cohniastreptothrix israeli* (Kruse, 1896)

2 *Ravaut and Pinoy's yellow actinomycosis*, found in France and caused by *Cohniastreptothrix thibiergei* Ravaut and Pinoy, 1909

3 *Acland's yellow actinomycosis* found in Europe, but the distribution of this form requires further investigation. It is caused by *Nocardia bovis* (Harz, 1877)

4 *Bouffard's yellow actinomycosis* found at Djibouti, in French Somaliland in the Anglo Egyptian Sudan, and in South West Africa and caused by *Nocardia somaliensis* (Brumpt, 1906)

5 *Krause's yellow actinomycosis*, found in Europe, and caused by *Nocardia krausei* (Chester, 1901)

6 *Garten's yellow actinomycosis* found in Europe, and caused by *Nocardia garteni* (Chester, 1901)

7 *Hesse's yellow actinomycosis*, found in Europe, and caused by *Nocardia liquefaciens* (Hesse, 1892)

8 *Chalmers and Christopherson's yellow actinomycosis*, found in the Anglo Egyptian Sudan, where it appears to be not uncommon, and caused by *Nocardia convoluta* Chalmers and Christopherson, 1916

9 *Eppinger's yellow actinomycosis* found in Europe, America (North and South) Asia, and Africa, and caused by *Nocardia asteroides* Eppinger, 1890

III *Red (sometimes yellowish) actinomycosis* Only one form of which is known —

Cc

This organism has red or yellowish grains which produce pinkish  
 on the ears (Plate VI Fig 8) and on potato tubers

ong to the genera  
 nged by cons der  
 like the Maduro  
 d the name of the

discoverer

They may be divided into —

1) black grains

## I THE BLACK ACTINOMYCETES

Only one variety found in Europe is known—viz *Babes and Mironescu's black actinomycosis* of which the fungus has never been classified

## II THE WHITE OR YELLOW ACTINOMYCETES

These may be differentiated by the characters of causal organisms into —

A Fungus difficult of cultivation grows best anaerobically  
 arthrospores absent—Genus 1 *Cohnistrepthothrix*

(a) Yellow grains —

1 *Israel's yellow actinomycosis* caused by *C israeli*  
 Krause 1896

(b) Very small white grains —

2 *Ravant and Pinot's yellow actinomycosis* caused by  
*C thubiergei*

B Fungus grows readily aerobically and produces arthrospores  
 —Genus 2 *Nocardia*

(a) Clubs present —

3 *Acland's yellow actinomycosis* caused by *N bovis*  
 Harz 1817

(b) Clubs absent

(c) Hard sheath around grains —

4 *Bouffard's yellow actinomycosis* caused by *N*  
*somalienensis* Brumpt 1906

(f) Hard sheath absent

(m) No growth on gelatine

5 *Krause's yellow actinomycosis* caused by *N*  
*krausei* Chester 1901

(n) Growth on gelatine

(o) Blood serum not liquefied —

6 *Eppinger's yellow actinomycosis* caused by *N*  
*asteroides* Eppinger 1899

- (f) Blood serum liquefied  
 (w) Pathogenic for laboratory animals —  
     7 *Garten's yellow actinomycosis* caused by *N. garteri*  
        Brumpt 1906  
 (x) Non pathogenic for laboratory animals  
 (y) Gelatine liquefied —  
     8 *Hesse's yellow actinomycosis* caused by *N. liquefaciens* Hesse 1892  
 (z) Gelatine not liquefied —  
     9 *Chalmers and Christopherson's yellow actinomycosis*  
        caused by *Nocardia contolula* Chalmers and  
        Christopherson 1916

### III THE RED (SOMETIMES YELLOWISH) ACTINO MYCOSIS

There is only one known variety—viz, *Carter's red* (sometimes yellowish) actinomycosis of which the causal organism is *Nocardia indica* (Kanthack 1893)

The reasons for believing that these are the causal organisms of the disease are the same as for maduromycosis and need not be repeated

1 A dense matrix 14

2 A number of irregularly shaped darker bodies the fungal masses embedded in the matrix

THE MATRIX—When the matrix is studied by the aid of higher magnifications it will be seen to be composed of white fibrous connective tissue containing a large number of connective tissue corpuscles and here and there a bloodvessel or a small group of bloodvessels which may or may not be associated with a collection

lumen

Connected with many of these vessels and often more or less

1. 2. 3. 4. 5. 6. 7. 8. 9. 10. 11. 12. 13. 14. 15. 16. 17. 18. 19. 20. 21. 22. 23. 24. 25. 26. 27. 28. 29. 30. 31. 32. 33. 34. 35. 36. 37. 38. 39. 40. 41. 42. 43. 44. 45. 46. 47. 48. 49. 50. 51. 52. 53. 54. 55. 56. 57. 58. 59. 60. 61. 62. 63. 64. 65. 66. 67. 68. 69. 70. 71. 72. 73. 74. 75. 76. 77. 78. 79. 80. 81. 82. 83. 84. 85. 86. 87. 88. 89. 90. 91. 92. 93. 94. 95. 96. 97. 98. 99. 100.

cytoplasm but when seen more correctly has a relatively fair quantity of cytoplasm in proportion to the size of the nucleus The nucleus being placed excentrically and the cytoplasm being

non granular and not eosinophile, this cell agrees with Unna's description of a healthy *plasma cell* as seen in actinomycosis

... of cell shows a large vesicular nucleus situate

termed 'fungai masses



FIG 848—ACTINOMYCOSIS

When a typical fungal mass is examined by means of a moderately high magnification, it can be seen to be composed of several distinct areas, which, working from the fibrous tissue matrix

4 *The Grain*—Situate in the cellular sheath there lies a more or less distinctly or indistinctly striated body, of varying shape and often with irregular edges, which is the grain, and is composed

observed to show collections of cells at intervals. These layers are composed of loose small cells, healthy and de-

cells may be remarked to be separated from the surrounding tissue by a little distance. These cells may be circumscribed by a new fungal growth, and so to speak, the formation of a new fungal mass.

that small areas of the tissue show signs of granular degeneration.

Another interesting feature but by no means confined to the fungal masses, is the presence of cells containing one or several, small or large, rounded eosinophile globules. These were called *fuchsin or Russell bodies*.

by Archibald (1911) thereof in Plates XV Report of the Khari the fungus and are

in cells at a distance from the fungus, in which case they aid in diagnosis as indicating the probable presence of a fungus somewhere. They are also seen in masses cut longitudinally and in the lymph spaces. They have been recorded by all workers

and other substances, a substance excreted by the fungus which only under certain conditions consolidates into the eosinophile form and into the clubs of certain species of *nocardia*.

**THE CELLULAR SHEATH**—All our observations tend to support Brumpt's view that primarily the fungus is enclosed in a cell which in the younger fungal areas near the older area is always multi-

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ss of its  
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## THE PARAMYCETOMAS

appear upon the scene and the fibrocellular coat begins to circumscribe the cells and the fungus while the damaged remains of the giant cell are seen retiring towards the periphery

Later the mononuclear cells mentioned above appear and these various cells together with detritus from the destruction of similar cells in a granular network form the cellular sheath of the specimens. This description although materially from a composite of Carter

Atta Brumpt and other body against different species of fungi

In more advanced cases the morbid anatomy is in maduromycosis with of course the difference in the grain

**Symptomatology, Diagnosis**—These are the same as for maduromycosis

**Treatment**—Vaccines have not given good results in our hands. The correct treatment is removal whenever possible but failing this iodide of potash in large doses may be administered

**Prophylaxis**—This is the same as for maduromycosis

## THE PARAMYCETOMAS

**Definition**—A paramycetoma is a disease which includes all growths and granulations producing enlargement deformity or destruction in any part of the tissues of man which are caused by fungi of any nature whatsoever but in which grains are either absent or so few and so small as to escape observation without prolonged search

**History**—In 1917 Chalmers and Archibald first proposed the differentiation and followed it up in 1918 by a further communication

**Clinical Remarks**—The Paramycetomas as already stated cannot be recognized without microscopic assistance because they present a varied group of clinical forms comprising chronic ulcers which may seem to be non malignant doubtfully malignant or malignant of growths which appear to be innocent or which are capable of diagnosis as doubtful carcinomata epitheliomata sarcomata or with reference to which no doubt is entertained the mind of the surgeon attending them

If removed in the more innocent forms or in the early stages they probably do not recur but in the later stages of the malignant forms they do recur but probably then as true malignant growths

As a diagnosis cannot be made clinically it behoves us to rely upon what it is to be based

**Diagnosis**—The recognition of a paramycetoma is based upon microscopical examination and consists in finding the following features—

- (a) Peculiar eosinophile bodies
- (b) Fungal filaments
- (c) Minute grains
- (d) Cultures and animal experiments
- (e) Minor points

(a) PECULIAR EOSINOPHILE BODIES—These are single bodies enclosed in cells or several large and apparently free bodies

In our opinion these bodies are composed of a chemical substance apparently formed in human tissues by several different kinds of

found in a paramycetoma is the *nocardial hypha*. These are easy of recognition to the trained eye but are apt to be mistaken by persons not acquainted with mycology and to be recognized as bacilli while their spores if present may be considered to be micrococci.

Other forms of fungi however may cause a paramycetoma—*e.g.* fungi of the type of a leptothrix.

It will thus be seen that just as we divided the mycetomas into the actinomycoses and the maduromycoses so can the paramycetomas be divided by the nature of the hyphal filaments into the

find and indeed are perhaps often absent when the case may be due entirely to hyphal filaments not collected into grains.

(d) CULTURES AND ANIMAL EXPERIMENTS—We have been un fortunate with our attempts at cultivation and in our animal inocu

(e) MINOR POINTS—Among minor points which are worth noting are the presence of many plasma cells either in good condition or degenerated.

The condition of the vessels which often show endarteritis or periarteritis just as in mycetoma is also worthy of note.

Another minor point is a peculiar glassy or vitreous macroscopic

These bodies if associated with many plasma cells degenerated plasma cells and changes in the bloodvessels and glassy degeneration are almost pathognomonic of the presence somewhere of a parasitic fungus.

The differentiation from mycetoma is not difficult as the grain is readily found in this growth which it certainly is not in a paramycetoma.

The differentiation from malignant growth is at the same time very easy and very difficult. Very easy because at once the specimen appears somewhat different from the typical malignant growth simulated and very difficult because it may require prolonged

removal the prognosis is good otherwise it is bad. If glandular excision is performed the presence or absence of infection is of the utmost prognostic value.

**Treatment**—The only known satisfactory treatment is the early and complete removal of the growth associated with glandular excision.

### THE PSEUDOMYCETOMAS

**Definition**—A pseudomycetoma resembles a mycetoma clinically in the presence of swelling, ulceration and discharge but differs therefrom in the absence of grains and from a paramycetoma in the absence of eosinophile bodies.

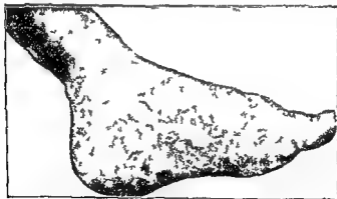


FIG. 849.—PSEUDOMYCETOMA OF FRAMBESIAL ORIGIN.

**History**—The name is used to indicate a peculiar type as clinically somewhat different from mycetoma by us in the second edition of this work.

**Remarks**—This condition is now well known to occur in the tertiary stage of frambesial tropica (yaws) and is not unfrequently seen in Ceylon. Breinl in New Guinea has described a similar



condition, known to the natives by the names 'roaki' 'buno' or 'auma' which he considers is a separate clinical entity from yaws. He says that the foot closely resembles Madura foot, without the presence of the typical grains in the pus.

A similar condition is known to occur in sporotrichosis. The

due to an angiohelminth in the foot of a negro in Brazil. The foot was much enlarged, with several nodules, from which white material exuded. Microscopical sections enabled a correct diagnosis to be made. The whole condition, however, resembled mycetoma, but neither grains nor fungal hyphæ could be found. His paper contains an excellent illustration.

### ACTINOMYCOSIS OF THE BODY.

Remarks.—The fungi producing Madura foot may occasionally invade other parts of the body instead of or in addition to the foot. They may attack the skin and deeper tissues of the hands, trunk, mammae in females, and also the deep organs, lungs, heart, liver, brain. For such conditions the term mycetoma is not applicable, but the term actinomycosis is used.

mycetoma)

Prognosis.—This depends greatly on the species of fungus causing the malady. The types due to *Nocardia bovis*, *Nocardia israeli*, or true actinomycosis, answer well to a potassium iodide treatment.

Diagnosis.—This is based on the presence of indolent or nodular masses breaking down with formation of pus, in which grains containing the fungus are found.

Treatment.—Potassium iodide in full doses (gr. xx) three or four times a day should always be administered.

#### Nodular Actinomycosis of Pinoy and Ravaut.

Historical and Geographical.—This condition and its fungus were studied by Pinoy and Ravaut in France.

Ætiology.—The condition is caused by a *Cohnstrepitothrix*. Only one case is

known described by Pinoy and Ravaut and due to *C. thibiergei* Pinoy and Ravaut, 1909 (see p. 1066).

**Symptomatology.**—In the only case on record there were numerous subcutaneous and intramuscular gummatous nodules which had developed very slowly they slowly softened some ulcerating. In the pus the fungus was found.

**Treatment.**—Potassium iodide is to be recommended.

Other nodular actinomycoses of nocardial origin, and characterized by the presence of abscesses or gummata in which fungi of the genus *Nocardia* are found, have been described by Riviere and others, and very ably in England by Foulerton,

#### Nocardial Abscesses.

Species of the genera *Nocardia* and *Cohniastreptothrix* may, at times, be found in the body. One such case is due to *Nocardia*.

#### TRENCH FOOT.

**Prophylaxis**—Trenches should be kept clean dry and sanitary as far as conditions permit. Officers and soldiers should receive instructions to keep the feet scrupulously clean and wear rubber boots in wet weather when possible. In our opinion putties should never be worn.

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## CHAPTER XCIV

# DERMATITIS VENENATA

Definition — Remarks — Historical — Climatology — Aetiology — Symptomatology — Diagnosis — Treatment — Prophylaxis — Varieties — Rhus group — Euphorbia group — Urtica group — Tectona group — Rue group — Buffalo bean group — Little-known group — Doubtful group — References

**Definition.**—The term *dermatitis venenata* includes a number of inflammatory skin lesions caused by the irritative action of poisonous principles contained in certain plants.

**Remarks.**—By the above definition it will be observed that the term *dermatitis venenata* is here used in the restricted sense of being only caused by plants leaving the dermatitis caused by animals to be treated in the chapter dealing with the Dermatozooses (p 220). Neither does the definition include *dermatitis medicamentosa* which is due to drugs administered internally or externally nor does it include *dermatitis factitia* which is caused artificially—e.g. beggars rubbing in *Ranunculus scleratus* Linnæus to produce sores for the purpose of inducing pity and the money usually associated therewith or recruits or soldiers utilizing various plants for purposes of malingering.

**Historical.**—From very ancient times it has been known that certain plants have stinging properties—e.g. many species of the genus *Urtica* of which *U. urens* Linnæus and *U. dioica* Linnæus are well known in Europe—but the effects of which are slight in comparison with the results produced by the species found in the East Indies—e.g. *U. urentissima* Comm. *U. crenulata* Roxburgh *U. stimulans* Linnæus and *U. ferox* Forster. One of the earliest Amœnitiatum which he refers

be found in books on travel on botany materia medica poisons as well as in textbooks on skin diseases. Thus in 1862 Van Hasselt made some references to the subject as did Bazin in the same year while Piffard in 1891 made many references to plants supposed to be causal agents.

In 1887 Whitl gathered the whole subject together in his work on *Dermatitis Venenata* and following this there was a leading article in the *Lancet* on the dermatitis produced by *Primula obconica*

Hance, 1880 In 1898 Blanchard made an excellent contribution with regard to *Arundo donax*, and was followed by Havard in 1899 Reynault in 1902, who detailed facts with regard to the disease as seen in Indo China, and by Wellman in 1907, who considered the

In 1914 Hornsey gave a good account of the fungus poisoning as seen in British North Borneo

In 1916 Vadala again referred to *Arundo donax* and in 1917 Chalmers and Pellala gave an account of a Sudanese dermatitis

distribution, it is a cosmopolitan complaint being found in all parts of the world It, however, requires further study in the tropics and it is for this reason that we have specially brought it forward in connection with skin diseases

**Ætiology.**—The causation of dermatitis venenata depends upon —

- I The plant
- II Personal idiosyncrasy
- III Confirmatory test
- IV The active principle

I *The Plant*—The following list largely compiled from White's book but altered so as to agree with our definition of dermatitis venenata and to include tropical plants gives those known to us to cause the complaint —

- Anacardiaceæ *Rhus venenata* De Candolle  
*Rhus toxicodendron* Linnæus  
*Rhus diversiloba* Engler  
*Rhus vernicifera* De Candolle  
*Semecarpus anacardium* Linnæus
- Ampelidaceæ *Cissus pruriens* Welwitsch
- Apocynaceæ *Nerium oleander* Linnæus
- Araceæ *Arisæma triphyllum* Schott  
*Symplocarpus fœtidus* Nutt

la Tour

- Compositæ *Erigeron canadense* Linnæus  
*Lappa officinalis*—*L. majus* Gaertner  
*Leucanthemum vulgare* Lambert  
*Solidago odora* Hooker and Arnold.

- Coniferæ *Abies excelsa* Link  
           *Juniperus virginiana* Thunberg  
           *Juniperus sabina* Linnæus  
 Connaraceæ *Thuja occidentalis* Linnæus  
               *Cnestis corniculatus* Lam  
 Crassulaceæ *Sedum acre* Linnæus  
 Euphorbiaceæ *Euphorbia corollata* Linnæus  
               *Hura crepitans* Linnæus  
               *Hura brasiliensis* Willdenow  
               *Hippomane mancinella* Linnæus  
               *Jatropha urens* Linnæus  
               *Stillingia sylvatica* Linnæus  
 Leguminosæ *Andira araroba* Aguiar  
               *Leucanthemum vulgare*  
               *Mucuna pruriens* De Candolle  
               *Stilobium stans* Kuntze  
 Linacææ *Linum utilissimum* Linnæus  
 Loasacææ *Mentelia oligosperma* Nuttall  
               *Mentelia lindleyi* Torrey and Gray  
 Loganiacææ *Gelsemium sempervirens* Aiton

This is a long list but all its members are not natives of the tropics in which the more important families are the Ampelidacææ the Anacardiææ the Apocyanacææ the Artocarpacææ the Con-

Hance 1880 In 1898 Blanchard made an excellent contribution with regard to *Arundo donax* and was followed by Hayard in 1899 Reynault in 1902 who detailed facts with regard to the disease as seen in Indo China and by Wellman in 1907 who considered the

In 1914 Hornsey gave a good account of the fungus poisoning as seen in British North Borneo

In 1916 Vadala again referred to *Arundo donax* and in 1917 Chalmers and Pekkola gave an account of a Sudanese dermatitis venenata caused by a member of the Rutaceæ

**Climatology**—With regard to *geographical distribution* it is a cosmopolitan complaint being found in all parts of the world. It however requires further study in the tropics and it is for this reason that we have specially brought it forward in connection with skin diseases

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*Rhus diversiloba* Engler  
*Rhus vernicifera* De Candolle  
*Semecarpus anacardium* Linnæus
- Ampelidaceæ: *Cissus pruriens* Welwitsch
- Apocynaceæ: *Nerium oleander* Linnæus
- Araceæ: *Arisæma triphyllum* Schott  
*Symplocarpus foetidus* Nutt
- Araliaceæ: *Aralia spinosa* Linnæus

a 1 our

- Compositæ: *Erigeron canadense* Linnæus  
*Lappa officinalis* = *L. major* Gaertner  
*Leucanthemum vulgare* Lambert

- Coniferæ *Abies excelsa* Link  
*Juniperus virginiana* Thunberg  
*Juniperus sabina* Linnæus.
- Cunraraceæ *Thuja occidentalis* Linnæus  
*Cnestis corniculatus* Lam
- Crassulacææ *Sedum acre* Linnæus
- Euphorbiacææ *Euphorbia mollata* Linnæus  
*Hura crepitans* Linnæus  
*Hura brasiliensis* Willdenow  
*Hippomane mancinella* Linnæus  
*Jatropha urens* Linnæus  
*Stillingia sylvatica* Linnæus
- Leguminosææ *Andira araroba* Aguiar  
*Leucanthemum vulgare*  
*Mucuna pruriens* De Candolle  
*Sisymbrium stans* Huntze
- Linacææ *Linum usitatissimum* Linnæus
- Loasacææ *Mentzelia oligosperma* Nuttall  
*Mentzelia lindleyi* Torrey and Gray
- Loganiacææ *Gelsemium sempervirens* Aiton
- Malvacææ *Malachra hirsuta* Huntze
- Orchidacææ *Cypripedium pubescens* Willdenow  
*C. pubescens* Salisbury
- Papaveracææ *Sanguinaria canadensis* Linnæus
- Phytolaccacææ *Phytolacca decandra* Linnæus
- Polygonacææ *Polygonum hydropiper* Linnæus  
*Polygonum acre* Hooker and Benth
- Ranunculacææ *Aconitum napellus* Linnæus

Rubiacææ

Rutacææ

Scrophulariacææ *Verbascum thapsus* Linnæus

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U

*Laportea canadensis* Gaudichaud Beaupré

Urticacææ Many species of *Urtica*.



naraceæ, the Euphorbiaceæ, the Leguminosæ, the Malvaceæ, the Rubiaceæ, the Rutaceæ, the Tropæolaceæ, and the Urticaceæ, the genera and species of which are indicated above

II *Personal Idiosyncrasy*.—When the poisonous principle is contained in the juice of cultivated plants, the poisoning is largely met with among gardeners florists, and people associated with plants in some way, when, however, the poisoning is due to a principle contained in some special hairs of a plant, whether cultivated or not, it is obvious that anyone may be affected; and when it is due to principles contained in the dust from dry wood, it is also obvious that carpenters and persons who cut or saw this wood will be most liable to be affected

With regard to the first series of cases, in our experience, there can be no doubt that some people are more liable to the affection than others, and it would appear that certain people suffering from any form of seborrhœa or allied condition, no matter how mild, are especially liable to be troubled by dermatitis venenata

As stated in the previous editions of this book we should not be surprised if, in the future, it will be found that certain forms of dermatitis venenata

III *Confirmatory Test*.—The crucial ætiological test is to remove the patient from the district in which the plant grows, to cure his

active principle

*Symptomatology*.—Sometimes the symptoms consist merely of itching, with or without an erythematous blush. At other times there may be marked erythema, with œdematous swelling in the affected part, which is often the face or the hands, or both. In more severe cases there may be present with papules, vesicles, and other symptoms as fever and

affected person may previously have been in excellent health. The termination is in quick recovery, especially if the causal agent is removed

*Diagnosis*.—The case presents the ordinary appearance of an acute dermatitis, and it requires patience and acumen to trace this to its correct cause

The characteristics of the disease are —

- 1 Acute dermatitis appearing suddenly and often without apparent cause in a previously healthy person
- 2 The history of the association with some plant by handling or being affected by the odour or even of being in the neighbourhood thereof
- 3 There may be history of previous similar attacks when in the vicinity of the suspected plant
- 4 The rapid recovery on removal from the causal plant

*dermatitis when the mite infests the plant*

In such a case the only possible method of diagnosis is to examine the plant and the patient carefully so as to exclude the presence of these insects and to reproduce the disease by means of a plant found to be quite free from mites

Treatment The most efficient prophylactic measure is to avoid contact with the plant

Strickler has tried in certain types the injection of minute doses of the poisonous principles extracted with absolute alcohol

**Prophylaxis**—When the patient knows that he is susceptible to the influence of a given plant care should be taken to avoid it

**Varieties**—Dermatitis venenata may be divided into several groups as follows—

- I
- II
- III
- IV
- V

- VI *Buffalo Bean Group*—Plants in which the causal agent is innumerable minute hairs which penetrate into the skin
- VII *The Little Kno en Group*—This is a group containing plants which apparently cause dermatitis venenata, but about which information is very defective
- VIII *The Doubtful Group*—This contains plants which probably do not produce dermatitis venenata, but perhaps harbour a mite which may be the causal agent.

with itching, eyes rise to papules, vesicles, and oozing-points on the inoculated limb, which in due course becomes brawny and pits on pressure. In a little time the swelling spreads to the eyelids and face, ears, lips, and nose, and great discomfort is experienced. In a

Another application of the active principle produced a relapse in forty minutes.

In the ordinary way the first attack is of slow development, the symptoms appearing some six weeks after the first handling of the wood, but a relapse is of rapid development.

The illness begins with the development of an acute inflammation of the skin of the hands, wrists, face, and neck, producing an appearance somewhat resembling erysipelas but without fever or concomitant sickness. The inflamed surfaces discharge, dry, and finally desquamate. The most suitable treatment according to Cash is to apply the unguentum glycerinus plumbi subacetates of the British Pharmacopœia every four hours, to administer bromides to allay the irritation and opium to relieve the laryngeal cough.

Other woods with similar properties are ebony (*Diospyros ebenum* Kon) found in Ceylon and India originally, and rosewood

r tree of  
sawn so

## V. THE RUE GROUP.

**HAPLOPHYLLUM DERMATITIS**—This has been described by Chalmers and Pekkola as occurring in the Anglo Egyptian Sudan

(Forsk.)  
ved to be  
usceptible  
the plant

at the original  
in was  
grew,  
The

experimental eruption (fig. 62) appeared in about 6 hours after rubbing the forearm for a few seconds with the leaves and flowers, which were partially dry after their long journey. The

of the ears, and eyelids became red and swollen, and there was much itching. Later the face became swollen, the lips cracked and oozed, and the lymph glands under the jaw and in the groin enlarged. All the symptoms rapidly disappeared when he ceased to work among these plants.



FIG. 850.—*Haplophyllum tuberculatum*

The treatment adopted was to wash the whole body after removal from the endemic area and to apply calamine lotion.



FIG. 851.—EXPERIMENTAL ERUPTION

**OTHER RUES**—According to Bentley and Trimen *Ruta graveolens* causes redness, swelling, and even vesication of the skin if much handled, while Le Maout and Decasne state that *R. montana* found in Spain produces erysipelatous-like conditions and ulcerating pustules on the hands of those who gather it.

## VI THE BUFFALO BEAN GROUP.

Letcher has written an interesting account of the sufferings produced by the minute hairs of the pods of the Buffalo bean. This bush with its silky green pods lives along the Luia River in Portuguese South Africa. The little hairs settle on the skin and set up violent irritation and burning lasting about one hour despite remedies. Natives plaster themselves all over with mud. This laconic description by no means expresses the views of sufferers who look upon the tree as one of the choicest creations of the devil.

## VII THE LITTLE-KNOWN GROUP

This group includes dermatitis due to —

- |                    |            |
|--------------------|------------|
| 1 Cashew nut       | 4 Oleander |
| 2 Cinchona quinine | 5 Rungus.  |
| 3 Nasturtium       | 6 Upas     |

•

upon the hands forearm

It is believed to be due

susceptible persons who should not be longer employed at this work. The rash disappears in two to four weeks after ceasing to

lacea) which  
sible persons

•

OLEANDER—*Nerium oleander* Linnæus (Apocyanaceæ) the oleander of Palestine and the East may in susceptible people cause symptoms like those produced by the Rhus group but this requires confirmation.

RUNGUS—This is a curious affection described in British North Borneo by Hornsey and is caused by contact with any part of the tree called rungus or ringus by the natives and said to be capable of being spread from a victim to uninfected persons. Within twenty four hours of handling the tree itching sensations and this is

The  
th the  
with a  
o foul

ulcers. Some people are immune. The condition heals naturally.

UPAS TREE—*Antiaris toxicaria* Lesch (Artocarpaceæ) according

THE DOUBTFUL GROUP

to Loudon causes cutaneous eruptions when wounded while Hasselt says that it affects the Javanese after the manner of the Rhus group when they come in contact with it This requires confirmation

VIII THE DOUBTFUL GROUP

The irritant poisoning caused by kayu rugas the juice of which produces painful bullae by kayu buta buta which causes violent dermatitis and conjunctivitis have not been well studied neither has the poisonous properties of the well known daffodil in fact the whole subject requires further study

CANE DERMATITIS—A peculiar dermatitis is found in people handling and cutting reeds (*Arundo donax* Linnæus) in Provence some parts of Italy some districts of Greece and other countries The dermatitis was studied by Blanchard and many observers among whom Thiebierge Berlese Brigi Aravandinos Siameni and Vadala may be mentioned It starts with severe itching and erysipelatoid eruption associated with the formation of blebs generally on the uncovered parts of the body but also on the genital organs which may become greatly oedematous and febrile symptoms with signs of coryza may appear After a few days the rash appears and is followed by desquamation

The Ætiology is doubtful Some authorities consider it to be due to the plant itself others to a fungus (*Ustilago hypodytes* Schlecht) and still others to a mite (*Aceria berlesii*)

Treatment consists in applying calamine and lead lotions

VANILLA DERMATITIS—This is often called Vanillismus and is due to *Vanilla planifolia* which is a native of Eastern Mexico but which also grows naturally in Tropical America and is cultivated in many parts of the tropics Workmen when handling the bean suffer from itching of the hands and face while the skin becomes covered with a pruriginous eruption and reddens swells and desquamates It would however appear that this is not due to the plant but to some mite thereon as it does not occur in sor works It is said that only persons with dry skins should work with vanilla, as the least drop of perspiration or moistening of the beans causes their destruction by fungal growth. The whole matter requires further investigation.

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London

CHAPTER XCV  
 ULCERATIONS

Cuta . . . . .

**CUTANEOUS LEISHMANIASIS (ORIENTAL SORE).**

**Synonyms.**—Delhi boil, Aleppo boil, Biskra boil, Bagdad boil  
 Ulcera de Bauru (Brazil), Bouton d'Orient, Ulcère d'Orient,  
 Chancro du Sahara, Dermite Ulcereuse Circonscrite (Corr.), Ende  
 mische Beulenkrankheit, Bottone d'Oriente Godownik, *ε*  
 'yearly boil (Caucasus), the Tartar name is 'Il jarassy' ('il' =  
 year, 'jarassy' = boil), Tschuban *ε*, 'yearly sore', Dous-el-  
 Kourmati *ε* 'date disease' (Turkish), Ghisud (Abyssinia);  
 Habb-es-Sanawi *ε*, 'yearly boil', Habb-es Sanah *ε*, 'boil of the  
 year', Bess-el Temur *ε*, 'date disease' (Arabian), Salek *ε*,  
 'annual' (Persian),  
*ε*, 'Afghan plague'  
 Taschkent Jarassy

MUCH TO HAVE BEEN THE FIRST TO SUGGEST THAT THE TRUE CAUSE MIGHT  
 POSSIBLY BE A PARASITE



Smith in 1868 and Fleming in 1873 claimed to have found eggs of a species of *Distoma* in the sections of specimens of the tissues derived from cases of Delhi boil. Carter in 1875 described a

the virus

id Boniet

In 1885

Cunningham described some parasites of a size and shape often endocellular staining with gentian violet (bodies as representing various mycetozoal parasite probably Monadidae

Riehl (1886) isolated a capsulated micrococcus Finkelstein and Chantemesse (1887) also cultivated a micrococcus similar to the organism described by Duclaux. Poncet in the same year described a coccus in sections and a very delicate bacillus. Le Dantec and Auché in 1894 found in a case of Biskra boil a streptococcus and the *Staphylococcus albus*. In 1897 Nicolle and Nourry Bey found a streptococcus which they believed to be specific. The organism was very slightly virulent. Attempts to inoculate monkeys with the disease did not succeed. In the same year Brocq and Veillon cultivated a streptothrix from a case of Aleppo boil. Crendiropuolo isolated in numerous cases a bacillus probably belonging to the *Proteus* group. Firth in 1891 stated that he had been able to confirm the presence of the Cunningham parasitic bodies in numerous cases of Delhi boil. He proposed for the parasite the name of *Sporoon furunculorum*. In 1898 Borowsky constantly observed in twenty cases of Sarten ulcer some peculiar organisms which he thought to be protozoa. In fresh preparations the bodies were very actively motile and presented a spherical shape, sometimes they were spindle shaped. The maximum diameter varied from 0.5 to 3  $\mu$ . The cell body stained very faintly. The nucleus was placed eccentrically. No chromatin bodies could be put in evidence. Schulgin in 1902 confirmed Borowsky's results and suggested that the disease might be conveyed by mosquitoes. In 1903 in a case of tropical ulcer occurring in a boy from Armenia Wright described bodies very similar to those found in cases of kala azar. These bodies may possibly be identical with those seen by Cunningham in 1885. Wright's discovery has been confirmed by Mesnil, Nicolle, James Strong, Plehn, Nattan Larrier, Splendore, Carini, Cardamatis, C. T. D. (in Arab. hold) and others who have Murzinowsky and they found similar in 1908. C. Nicolle in 1908. In the same year and 1910 C. Nicolle and others produced the disease in monkeys and dogs and in 1913 to 1914 Gonder, Row and Laveran infected mice and other rodents. In 1917 Laveran

published a most useful and complete treatise on the malady and other leishmaniases

**Geographical Distribution.**—The disease is endemic in many tropical and subtropical regions. It is found also in temperate zones. In Africa it is found in Morocco, Tunis, Tripoli, Algeria, and Sahara (Biskra Gafsa), Egypt, Sudan Congo, West and East

Larava and others from Italy. It is known also in Brazil and other countries of South America and in French and British Guiana, where it is known as Pian bois or forest yaws and has been observed by Darling in Panama so that the name 'Oriental sore' is somewhat misleading.

It is generally much more common in large towns than in the country. In some cities it is so prevalent that even visitors of a few days only may not escape it. Its occurrence appears to be influenced by the seasons as according to Hirsch in the tropics it is most prevalent at the beginning of the cool season and in more temperate climates at the end of summer. Laveran says that in Biskra from September to October inclusive, the slightest wound tends to become transformed into the 'bouton'. In some years it has been found to be more prevalent than in others.

A peculiarity of the geographical distribution of the disease

Dull etc.

**Ætiology.**—The disease is caused by *Leishmania tropica* Wright,

It is produced cutaneous lesions in monkeys but such lesions differ histologically from true Oriental sore.

Mesnil Nicolle, and Rembarger have observed the parasites to be present occasionally in true polymorphonuclear leucocytes, besides being found in the mononuclear leucocytes as usual. Mesnil has observed in some cases a typical 'bacillary' form.

Smith in 1868 and Fleming in 1873 claimed to have found eggs of a species of *Distoma* in the sections of specimens of the tissues derived from cases of Delhi boil. Carter in 1875 described a

staining with gentian violet. Cunningham inclined to regard these bodies as representing various stages of the development of a mycetozoal parasite probably belonging to the group of the Monadidæ.

Riehl (1886) isolated a capsulated micrococcus. Finkelstein and Chantemesse (1887) also cultivated a micrococcus similar to the organism described by Duclaux. Poncet in the same year described a coccus in sections and a very delicate bacillus. Le Dantec and Auché in 1894 found in a case of Biskra boil a streptococcus and the *Staphylococcus albus*. In 1897 Nicolle and Nourry Bey found a streptococcus which they believed to be specific. The organism was very slightly virulent. Attempts to inoculate monkeys with the disease did not succeed. In the same year Brocq and Veillon cultivated a streptothrix from a case of Aleppo boil. Crendiropuolo isolated in numerous cases a bacillus probably belonging to the Proteus group. Firth in 1897 stated that he had been able to confirm the presence of the Cunningham parasitic bodies in numerous cases of Delhi boil. He proposed for the parasite the name of *Sporozoon furunculiformis*. In 1898 Borowsky constantly observed in twenty cases of Sarten ulcer some peculiar organisms which he thought to be protozoa. In fresh preparations the bodies were very actively motile and presented a spherical shape, sometimes they were spindle shaped. The maximum diameter varied from 0.5 to 3  $\mu$ . The cell body stained very faintly. The nucleus was placed eccentrically. No chromatin bodies could be put in evidence. Schulgin in 1902 confirmed Borowsky's results and suggested that the disease might be conveyed by mosquitoes. In 1903 in a case of tropical ulcer occurring in a boy from Armenia Wright described bodies very similar to those found in cases of kala azar. These bodies may possibly be identical with those seen by Cunningham in 1885. Wright's discovery has been confirmed by Mesnil, Nicolle, James Strong, Plehn, Nattan-Larrier, Splendore, Curini, Cardamatis, Wenyon, Gabbri, Lacava, Balfour, Archibald and others who have greatly extended our knowledge of the disease. Marzinowsky and Bogrow state that independently from Wright they found similar bodies in cases of Pendjeh ulcer from Persia. In 1908 C. Nicolle and A. Sicre succeeded in cultivating the organism. In the same year and 1910 C. Nicolle and his co-workers reproduced the disease in monkeys and dogs and in 1913 to 1914 Gonder, Row and Laveran infected mice and other rodents. In 1917 Laveran

published a most useful and complete treatise on the malady and other leishmaniasis

**Geographical Distribution.**—The disease is endemic in many tropical and subtropical regions. It is found also in temperate zones. In Africa it is found in Morocco Tunis Tripoli Algeria and Sahara (Biskra Gafsa) Egypt Sudan Congo West and East

Cyprus Crete and Greece Cases have been reported by Gabbi Lacava and others from Italy It is known also in Brazil and

few days only may not escape it Its occurrence appears to be influenced by the seasons as according to Hirsch in the tropics it is most prevalent at the beginning of the cool season and in more temperate climates at the end of summer Laveran says that in Biskra from September to October inclusive, the slightest wound tends to become transformed into the bouton In some years it has been found to be more prevalent than in others

A peculiarity of the geographical distribution of the disease

boil etc

**Etiology**—The disease is caused by *Leishmania tropica* Wright

description of *L. tropica* and its varieties is found in Chapter XIX p 378 Nicolle and Manceaux Laveran and others have occasionally succeeded in producing in monkeys and dogs Oriental sore by inoculation of cultures Row by inoculating cultures of *L. donovani* has produced cutaneous lesions in monkeys but such lesions differ histologically from true Oriental sore

Mesnil Nicolle and Remlinger have observed the parasites to be present occasionally in true polymorphonuclear leucocytes besides being found in the mononuclear leucocytes as usual Mesnil has observed in some cases a typical bacillary form.

similar at the very beginning from mosquito bites for which they are often taken by the patients. The spots however instead of slowly fading become red and shotty with an inflamed areola which later becomes markedly indurated. The papules slowly

generally not exceeding three to four months the ulceration of the nodule begins. At first the ulceration is very superficial and



FIG. 853.—ORIENTAL SORE IN A PERSIAN SOLDIER  
(From a photograph of Drs. A. Bussière and Nattan-Lattier.)

exudes a  
darkish  
disintegrat-

tissues surrounding the ulcers may become oedematous. If later the darkish scab be removed an ulcer is seen about an inch or

and adherent  
process and  
spread. The  
indus  
ilcer  
urg  
here

is a secondary pyogenic infection. The examination of the blood

break down again.

Oriental sore may be single or multiple. Two or three are frequently found on the same patient, but occasionally there are many

palms, soles or scalp. The affection attacks people of any race

as long as five months, and Wenyon, who inoculated himself, ob-

themselves experimentally. Attacks of irregular fever during the incubation and the course have been recorded by several other authors. During one of these febrile attacks Neumann observed *Leishmania tropica* free in the liquor sanguinis of the patient.

The duration of the eruption varies from four or five months to twelve months and more. Relapses may occur, but true reinfections are rare.

**Clinical Varieties**—The following clinical varieties may be distinguished—

1. The common variety—*Oriental sore sensu stricto*—to which the description given above refers. This variety which is the usual type met with in Asia, Africa, and South Europe while comparatively rare in America is characterized by the presence of one or several nodules which slowly ulcerate with or without symptoms of general infection such as fever and enlargement of the spleen. Very rarely, in addition to the cutaneous nodules there may be ulcerative lesions on the mucosa of the mouth and nose as noted by Cardamatis in Greece and Lacava and Gabbi in Italy.

an  
pe

by  
of  
Fc

and Brumpt created a new species for the leishmania found in the

—*L. nilotica* Brumpt, 1913

4 The *frambesiiform* variety This is characterized by the pre



FIG 854—FOREST YAWS  
(From a photograph by Sambon)

6 The *deep ulcerative* variety This is found in South America and is characterized in most cases by the presence on various parts of the body of deep large ulcers running a very long course and with practically no tendency to spontaneous cure. In some cases the condition is localized to the ear, which may become perforated (*oreya de* by Low and is du

Diagnosis.—The diagnosis of all types of cutaneous leishmaniasis is made with absolute certainty only by examining microscopically the

Amazon natives with a word which means *sponge*. The natives believe that the same affection attacks horses, mules, and donkeys.

tendency to spontaneous cure. It is probably due to a species of leishmania biologically different from *L. tropica* and most authorities consider it to be due to *L. tropica* var *americana*, the variety of leishmania which is the cause of espundia (see below).

2 The few eruptive elements—often one single element—situated as a rule on uncovered parts of the body

3 The course a small papule which slowly enlarges into an indurated nodule indolent smooth or slightly scaly and after

patients who after the general eruption has disappeared have

definite diagnosis in difficult cases is the microscopical examination. To do this the scab is removed and a scraping is taken from the floor and edges of the ulcer. The preparation is then coloured with Leishman's or Giemsa's stain or any other of the numerous modifications of Romanowsky's method and examined for the presence of *Leishmania tropica*. The search must be prolonged in some cases as the parasites may be very rare.

**Prognosis.**—In the common type the prognosis is good *quoad vitam*. Very occasionally the disease may end fatally owing to the ulcers becoming phagedæmic and to secondary septicæmic and

Occasionally a prolonged patient may complain of tachycardia has been noticed and a few cases of sudden death have been ascribed



to the action of the drug which may produce a severe fatty degeneration of the heart liver and kidneys

**TARTAR EMETIC CARBOLIC SOLUTION**—This contains 1 per cent tartar emetic and  $\frac{1}{2}$  per cent carbolic acid and does not need to be sterilized or  
*Re. Knafel filter*

useful

The solution is prepared in bulk in a sterile bottle and tested for sterility forty eight hours after preparation it may then be put up in small 1 c c ampoules which it is advisable to keep in a cool dark place The presence of carbolic acid decreases the pain induced by tartar emetic and makes the solution sterile

Tartar emetic gr viii ac carbolic ℥ss

now has  
percent  
solution  
der but

sore was  
re acid

oil of  
vaseline  
gr v to  
c ac d) s

app u

Salvarsan and atoxyl have been used without any benefit  
 Marznowsky after removing the crust and cleansing the ulcer with antiseptic lotion applies a 10 per cent lotion of ferropyrin to stop the bleeding

drying up

carbolic acid.

acid 5 per cent, or tincture of iodine

### MUCO-CUTANEOUS LEISHMANIASIS (ESPUNDIA).

*Synonymy.* Mucocutaneous Leishmaniasis. Uta. Chancro Espundia.

America since time immemorial. According to Tamayo espundia lesions are described on certain water crops of the ancient Yucas

animals by inoculating pure cultures of the organism. Breda's work was confirmed by Verrotti and De Amicis. The term 'boubas,'

to the action of the drug which may produce a severe fatty degeneration of the heart liver and kidneys

TARTAR, EMETIC CARBOLIC SOLUTION—This contains  $\frac{1}{2}$  per cent tartar

useful

sterility  
all i c c  
sence of  
akes the

solution sterile

Formula No 2 (Castellans)—Tartar emetic gr viii ac carbolic ℥x

decreases the emetic action of the drug

LOCAL APPLICATIONS OF ANTIMONIAL PREPARATIONS—G C Low has

$\mathfrak{z}$ i iodoform or eucrophen gr v ung ac bonic  $\mathfrak{z}$ i balsam Peru gr v to vaseline  $\mathfrak{z}$ i) or an antiseptic powder (iodoform or xeroform or bonic ac d) is applied

Salvarsan and atoxyl have been used without any good result Marzinowsky after removing the crust and cleansing the ulcer with antiseptic lotion applies a 10 per cent lotion of ferropyrin to stop the bleeding

daging up

### MUCO CUTANEOUS LEISHMANIASIS (ESPUNDIA)

Synonyms—Naso oral Leishmaniasis Uta Chancre Espundique

**Definition**—A chronic ulcero granulomatous affection of the skin and mucosa of the mouth and nose due to *Leishmania tropica* Wright 1903 var *americana* Laveran and Nattan Larrier 1912

**Historical**—Espundia seems to have been present in South America since time immemorial. According to Tamayo espundiales are depicted on certain water vases of the ancient Incas. The malady has been known to local medical men for many years in

animals by inoculating pure cultures of the organism. Breda's work was confirmed by Verrotti and De Amicis. The term boubas

to the action of the drug, which may produce a severe fatty degeneration of the heart, liver and kidneys

**TARTAR EMETIC CARBOLIC SOLUTION**—This contains 2 per cent tartar emetic and 1 per cent carbolic acid and is used as a local application

useful.

inferior to that of tartar emetic

Salvarsan and atoxyl have been used without any good result  
Marzinowsky, after removing the crust and cleansing the ulcer with antiseptic lotion, applies a 10 per cent lotion of ferropyrin to stop the bleeding

daring up

### MUCO-CUTANEOUS LEISHMANIASIS (ESPUNDIA)

**Synonyms**—Naso oral Leishmaniasis Uta Chancre Espundique d'Escomel (Laveran and Nattan Larrier) Leishmaniasis cancerosa (A da Matta) Bubas Braziliانا (Breda) Smith's disease Breda's disease Bueno de Miranda and Splendore's Leishmaniasis American Leishmaniasis (Laveran and Nattan Larrier)

**Definition**—A chronic ulcero granulomatous affection of the skin and mucosa of the mouth and nose due to *Leishmania tropica* Wright, 1903 var *americana* Laveran and Nattan Larrier 1912

**Historical**—Espundia seems to have been present in South America since time immemorial. According to Tamayo espundia

used by Breda was a rather unfortunate one as it led to much confusion this term being generally used by tropical authors as a synonym for frambœsia while the natives as noted by Splendore and others use it indiscriminately to indicate various ulcerative lesions of widely different nature.

Escamél in 1691 gave a very good description of espundia as



FIG 855—ESPUNDIA  
(After Splendore)

usually deeper than *L. tropica*; Laveran  
have made an

nucleus being flattened have created a  
new variety *L. tropica* Wright 1903  
var *americana* Laveran and Nattan  
Larrier 1912 Vianna had previously  
created a new species *L. brasiliensis* but  
this species has not been generally  
accepted More recent important

has been carried out by Horta  
da  
Brues  
Christopherson and many  
others

**Geographical Distribution**—The disease has been reported from  
Peru Brazil Paraguay Argentina Colombia and other parts of  
South America In Brazil it is especially common in the regions

Sandia and close to the river It has been found in the  
by Christopherson



FIG. 856—ESPUNDIA  
(After Splendore)

Splendore has occasionally seen giant cells no cell nests have been observed. The stroma consists of some fibrils which are stained with difficulty. The lesions are not very vascular.

**Communicability.**—The infection may be conveyed by direct contact from person to person the virus being absorbed through some abraded surface fissure or small wound. It may also be

As regards the *reservoir* of the virus certain observers suspect dogs in which occasionally a similar or identical affection to the human one may be found. In Paraguay there is a popular belief that the rattle snakes are the reservoir of the virus.

Darling and Townsend have brought forward the hypothesis that the



**Symptomatology**—The incubation period is unknown. The malady generally begins with a nodule on some uncovered part of the body which fairly quickly breaks down and an ulcer is formed. This first lesion is called by Escamel *espundial chancre*. It is mostly found on the face. The



FIG. 93.—ESPEYDIA A SIDA  
CASE

(From a photograph by  
Christopherson.)

granulating and there is abundant purulent secretion which dries up forming thick crusts. The ulcer after some months or even one or two years heals up leaving a thick scar. While this primary ulcer is still open but oftener after it has healed the characteristic lesions occur on the mucosa of the mouth and nose with or without the appearance of further ulcerative lesions on the skin of various regions of the body. The lesions on the mucosa of the mouth are ulcero-granulomatous, often frambesiform, and may invade the hard and soft palate, the gums, the labial mucosa. They may form on the palate a diffuse granular mass with deep furrows. The mucosa of the nose is very often attacked and destruction of the cartilages may take place, causing a marked deformation of the nose. Alfredo Martins observed that the bones are

destroyed, the skin is often redematous, and patches of hard edema may be found below the eye. The pathological process may extend to the pharynx and larynx. In some cases the patient may become aphonic and complain of great pain during swallowing. His breath may be very offensive. There may be no fever.

The course is chronic. The disease may last for twenty to thirty years, death being generally due to some intercurrent disease. In a few cases the affection may be self-limiting, either than the nasal and paranasal. For instance, a case of nasal tuberculosis

has been recorded by Alfr da Matta. In the last stage the patient becomes cachectic, and his appearance may be that of a carcinomatous patient.

**Prognosis.**—Before the introduction of tartar emetic by Vianna the prognosis used to be very bad though occasionally when the diagnosis was made as soon as the primary cutaneous lesion appeared, and this was destroyed the further progress of the disease was prevented.

presence of a leishmania and absence of yeast like or monilia like fungi. From syphilis it can be diagnosed by the uselessness of the mercurial and salvarsan treatment from morva by the absence of

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According to Escome! if the primary lesion be excised or destroyed the further course of the disease is prevented.

**Prophylaxis.**—Abrasions fissures and any ordinary traumatic small sores should be kept well disinfected and protected with antiseptic dressings to prevent infection with the espundia virus. Any insect bite should immediately be touched with tincture of iodine.

#### INDIAN ORO-PHARYNGEAL LEISHMANIASIS.

**Remarks.**—It may be of interest to give a brief account of an ulcerative condition of the throat observed by one of us in two Europeans who had long been living in India.

**Etiology.**—In one of the two cases observed, scrapings from the ulcers contained typical leishmania bodies very similar or identical to *Leishmania tropica* and *L. donovani*. In the other—which was

The important researches of Keysseltz and Mayer and the more recent ones of Wolbach and Todd confirm Prowazek's work.

distinguishes also male and female forms. Various shaped spirochetes

*Inoculation Experiments—Communicability*—Experiments to inoculate the disease in men and in the lower animals have been made by several authors. Blaise inoculated himself with the secretion of a case of *ulcus tropicum* but no ulcer was produced—only a

berstadter tried to reproduce the affection in monkeys (orang-outang and *Macacus cynomolgus*) but without success.

It would seem therefore that to a certain extent the disease is not directly contagious. It is probable that some insects or other blood sucking vermin may play an important role in the transmission of the disease. In Ceylon patients often state that the ulcer developed at the site of a leech bite. Leeches are extremely common in Ceylon and other tropical countries. Prowazek in Java has examined many leeches but he never found any spirochetes. The blood of the leech he examined was quite free from spirochetes. He examined very actively and found only *Spirillum* and *Spirillum* very mobile spirochete.

in dry or higher regions. It may be that it is in hot climates that the carriers of the infection thrive.

The disease is very common among the poorer classes of the population who go barefooted and wear but scanty clothes. We

have observed it very often in beggars and in scabies patients. The disease is much more common in adults than in children in men than in women.

**Histopathology**—This has been thoroughly studied by Keysseltz and Mayer and by Wolbach and Todd. The surface of the ulcer is covered often by a tenacious membrane composed almost solely of coarse meshed hyaline fibrin with detritus and masses of sprochetes and various bacteria. The fundus

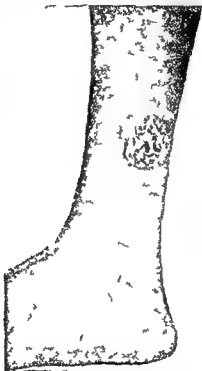


FIG 858—ULCUS TROPICUM TYPICAL

and walls consist of granulation tissue which does not present any characteristic feature. The deeper tissues and corium are surrounded by a

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It can be seen that the superficial layers of the fundus show a large amount of granular detritus and numerous foci of leucocytic infiltration while the

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of the body. It is single in most cases but two or more ulcers may be found in some patients.

Ulcus tropicum begins with the appearance of a small painful occasionally pruriginous papule or papulo-pustule surrounded by a deeply infiltrated dusky red areola. The initial lesion soon undergoes purulent and degenerative changes which rapidly extend to the infiltrated area. A sloughing process sets in and an ulcer



FIG 839—ULCUS TROPICUM EARLY STAGE

is formed which gradually extends in depth and surface. The margins are not sensibly raised nor thickened unless the case be very old. They are not perpendicularly cut nor undermined as a rule the whole ulceration having generally a roundish or oval outline and when the secretion is removed a concave fundus. The parts surrounding the ulcers are often oedematous and somewhat painful on pressure. It is remarkable however how comparatively little pain there is in many cases.

When the patients are first seen the whole ulcer is generally covered with a thickish dirty greyish secretion exhaling a highly offensive odour. On removing the secretion the fundus will be found to be of a red colour or in chronic cases pale pinkish and feebly granulating.

The fundus is often somewhat infundibular in its central area and not rarely may present a circular raised ridge which divides

— 6 — one and

deeper structures—muscles tendons and periosteum are affected  
 phage till the  
 The course is always chronic lasting for months—in fact the  
 ulcer has hardly any tendency to spontaneous healing if untreated  
 Healing takes place by a very slow process of granulation and  
 happens from the periphery A thick whitish often disfiguring  
 on the

The microscopic examination of the greyish bad smelling secretion shows  
 leucocytes undergoing various degenerations some red blood cells threads  
 of connective tissue and very  
 often spirochaetes and fusiform  
 bacteria of various types some  
 times accompanied by the usual  
 pyogenic cocci In old untreated  
 cases larvae of flies may be found  
 small acarids and ants

Diagnosis—According to  
 Le Dantec Vincent and  
 several other observers ulcus  
 tropicum is identical with  
 hospital phagedæna The  
 fact however that in con-  
 trast to hospital phage-  
 dæna ulcus tropicum shows  
 very little or no direct con-  
 tagiousness and in most  
 cases is self limited clearly  
 shows in our opinion that



FIG. 860.—ULCUS TROPICUM

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 perience

however the reverse is much more common—viz an ulcus  
 tropicum becomes infected with frambœsia virus takes a papil-  
 followed by a general eruption of

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*Oriental Sore*—An old standing Oriental sore may present some characters of a chronic *ulcus tropicum* though generally a tropical ulcer is much larger. The process of ulceration and breaking down is very slow in Oriental sore while it is very rapid in *ulcus tropicum*. In difficult cases the search for *Leishmania tropica* which is present in Oriental sore will clear the diagnosis.

*Ulcus Cruris Varicosum*—There are often varicose veins visible the parts surrounding the ulcer are congested and frequently eczematous the ulcer is often shallow and irregular.

*Ulcers of Tubercular Origin*—They are rare on the lower extremities and their development is long and insidious. In *ulcus*

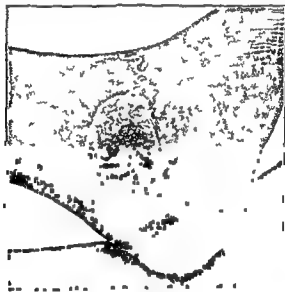


FIG. 861.—ULCUS TROPICUM

*tropicum* the tubercular cuti and ophthalmic reactions are negative  
ur

all sporotrichosis often show at first the characters of gummatoid. The bacteriological examination will reveal the presence of the fungi.

*Acladiosis*—The ulcerative lesions are multiple and of smaller dimensions. The bacteriological examination will reveal the presence of *Acladium castellanii* Pinoy.

*Mycosis Fungoides*—Is generally preceded by a general pruriginous dermatitis of various character lichen planus like psoriasis.

like eczematous like The ulcerative lesions are multiple and have the characters of granulomata

**Prognosis**—If untreated tropical ulcer has very little or no tendency to spontaneous recovery and in some cases may extend damaging the deeper structures tendons muscles nerves and vessels Occasionally a general septicæmia and pyæmia may supervene In countries where frambœsia is endemic the ulcer often gets infected with its virus and the patient develops a general eruption of frambœsia

**Treatment**—Salvarsan neosalvarsan and their substitutes have been administered by intravenous or intramuscular injection by Werner Hallenberger and others with success in certain cases

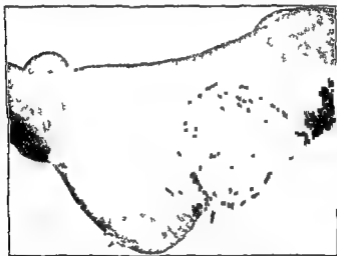


FIG 862.—ULCUS TROPICUM WITH CORNU CUTANEUM

The details of the treatment are found in the chapter on Frambœsia (p 1260) Mercury and potassium and sodium iodides are useless but calcium iodide (gr iii) well diluted three times daily seems occasionally to be of some slight benefit As regards local

kept at rest the  
by using a disin-  
1 in 1000 cyllin  
permanganate of

potash 1 in 7000

For the first few days it is better not to apply any so called disinfectant ointments or powders simply keep the ulcer covered with gauze moistened as often as possible with one of the disinfecting solutions already mentioned This generally stops the



formation of the greyish dirty secretion. The ulcer will then appear clean and of a pinkish colour, but whatever be the further treatment used whether powders (iodoform boracic acid) or disinfecting ointments (white red precipitate or iodoform ointments) the improvement will be very slow and several weeks and often months will elapse before a firm cicatrix is formed. Much quicker results will be obtained by using a protargol ointment. The ulcer is cleaned every morning with a perchloride lotion (1 in 1000) then a protargol ointment (5 to 10 or 20 per cent) is thickly spread on a piece of lint or gauze and applied to the ulcer which is then fairly firmly bandaged.

The superiority of the protargol treatment over other kinds of local treatment is patent in many cases. Castellani who introduced it for *ulcus tropicum* made the following experiment in a patient presenting two ulcers of little

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of 10 parts of

### ULCUS INTERDIGITALE

This affection is not rare among natives. It was described in 1909 by Castellani whose work has been recently confirmed by Breinl, Martinez and Lopez. The patient complains of some itching between the toes though no papules or vesicles are seen. After a few days a fissure develops and enlarges into a large oval ulcer with irregular margins. There is no pain. The ulcer is generally very shallow and does not show signs of depth. It heals in a few days if dressed twice daily with a bismuth boric acid ointment —

|                    |        |
|--------------------|--------|
| Bismuthi subnitrat | gr xxx |
| Acidi borici       | gr xv  |
| Vaseline           | ʒi     |

### ULCUS INFANTUM

Under this name Castellani describes a disease which he first met with in his researches on the island of Ceylon. It is common in Ceylon and India.

and Savana in the East and North Africa.

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shows ■ yellowish  
n, and ■ small ulcer  
red fundus The  
which dries into a



FIG 863.—ULCUS INFANTUM

yellow crust. If after some days the crust is removed, the ulcer will be found much larger and deeper—the size of a shilling to a half-crown piece. The ulcer is generally indolent, except on pressure. There may be a little pruritus. The ulcer may be single or multiple. The inguinal lymphatic glands may become enlarged, and occasionally the child has fever. The duration is between four to six weeks and three or four months. On healing, a permanent whitish scar is left.

**Diagnosis**—The *ulcus infantum* is differentiated from *ulcus tropicum* by the less severe symptoms by being almost always multiple by the smaller dimensions of the sore by the absence of spirochaetes and by the absence of any tendency to phagedæna. In contrast to veldt sore the ulcers are deep and the crust very thick. No streptococcus is found.

**Treatment**—Touch the ulcers with pure hydrogen peroxide once every other day and dress them with simple boric acid lotion (2 per cent.)

### REMARKS ON ULCERS.

Ulcerative conditions of the skin are extremely common in the tropics. They may be classified as follows—

- 1 Cutaneous leishmaniasis
- 2 *Ulcus tropicum*
- 3 *Ulcus infantum*
- 4 Veldt sore
- 5 *Ulcus interdigitale* <sup>1</sup>
- 6 Gangosa ulcers
- 7 Leprotic ulcers
- 8 Ulcers of framboeal origin
- 9 Elephantoid ulcers
- 10 Blastomycetic sporotrichitic acladiotic and other ally hyphomycetic ulcers
- 11 Cancerous and sarcomatous ulcers
- 12 Tubercular ulcers
- 13 Syphilitic ulcers
- 14 Glanders ulcerations
- 15 Ulcers of pyogenic origin (*pyosis tropica*)
- 16 Ulcers due to varicose veins
- 17 Undetermined chronic or subchronic ulcerations

The ulcerative conditions which may be considered as strictly

present enormous dimensions and may show secondary infection and become phagedæmic. Ulcers due to varicose veins are very common among rickshaw coolies who have to run and stand for hours at a time. It is remarkable how quickly they heal in most cases in these coolies if the patient is kept at rest for some time whereas in temperate zones the healing of varicose veins ulcers is of very long duration.

### Undetermined Subchronic and Chronic Ulcers

Knowledge of this group of ulcers is scanty but the investigations of Strong Stitt Rho Wherry and Clegg and others have thrown some light on this subject. Our experience tallies with that of Stitt and we therefore consider that such ulcers may be roughly divided into three groups —

- 1 Septic Ulcers—Ulcerations following on Neglected Wounds.
- 2 Painless Chronic Ulcers
- 3 Diphtheroid Ulcers

**Septic Ulcers**—Ulcerations following on Neglected Wounds—  
These are of pyogenic origin and often very large dimensions

The opsonic treatment also gives good results

Nichols has called attention to discharging sores in the Philippine Islands called *puente* which are produced by the natives applying some lime to the skin and afterwards betel powder with the object of counter irritation

**Painless Chronic Ulcers**—A small red scaly slightly itching spot appears generally on the legs and gradually enlarges for about four to eight weeks when the affected area begins to exude a serum which quickly dries into crusts. Under the crust ulceration slowly takes place. At first the ulcers are shallow and may have undermined edges later they are often punched out and may become indurated. There is no pain except slight pain on pressure and a pale cicatrix

a prevalence of mononuclear cells polymorphonuclears being practically absent. No pyogenic organisms are found

**Treatment**—The treatment is difficult. Cauterization does very little. In some cases the application of bismuth subnitrate xeroform novoforn dermatol and firm bandaging is useful. In others a protargol ointment (5 to 10 per cent) or a nitrate of silver ( $\frac{1}{2}$  per cent) balsam of Peru (1 per cent) ointment is of advantage. Allantoin preparations may also be used. When the ulcers are

be found covered with greenish pus. The membrane reforms rapidly and apart from the dark colour it closely resembles diphtheria membrane. These ulcers extend rapidly but do not take as a rule a true phagedænic character. The margins after some time may become indurated but do not show a punched appearance. Scrapings taken from the fundus show numerous these ulcers aicum except I

**Treatment**—Excision is not to be advised as in Stitt's and own experience when this has been done additional lesions have appeared. Bier's passive congestion method is painful and does not improve the condition. On the whole the best treatment is to keep the ulcers well disinfected with a perchloride lotion (1:2000) occasionally touching them with pure hydrogen peroxid. Should the ulcers become phagedænic the application of pure carbolic is advisable.

### GRANULOMA INGUINALE

**Synonyms**—Ulcerating granuloma of the pudenda. Granuloma Venereum (Brooke). Esthiomene de la vulve.

**Definition**—Granuloma inguinale is a chronic granulomatous affection of probable protozoal origin attacking the generative organs from which it spreads to the inguinal regions and the perineum.

**History**—In 1896 Conyers and Daniels described a disease of the generative organs in both men and women in British Guiana which was very painful, disfiguring and contagious. Daniels thinks that it was previously described by Macleod and Martin in India. Since then papers have appeared on the subject by Ozzard, Galloway, Wise, Donovan, Siebert, Flu, Martin, Gabb, Sabella, Torres, Rabello, Pijper, Mayer, Newham and Low and many others.

**Climatology**—It occurs in British Guiana, the West Indian Islands, West Africa, South Africa, India, South China and Northern Australia, but is rare in Ceylon, Malaya, Sudan and Central Africa. by Gabb and been met with

**Ætiology**—It appears in the genitalia of both sexes after puberty but is rare after forty-five years of age.

Donovan in 1905 described certain peculiar rod-like bodies  $2\mu$  by  $1\mu$  lying singly or in groups in mononuclear cells obtained by scraping the sores. Donovan stated that the bodies looked like

Markham Carter in 1910 described the parasites as bean shaped bodies resembling the gregariniform stage of a herpetomonas or a crithidium and came to the conclusion that the affection was due to either a herpetomonas or a crithidium.

Flu in 1911 in South America confirmed Siebert's work but considered the bodies to be bacilli with capsules and not cocci. At the same time however he called attention to the possibility of the bodies being a stage of a chlamydozoal infection. Martini in 1913 announced that he had succeeded in cultivating the germs described by Siebert and Flu on blood agar. He described them as anaerobic capsulated Gram negative diplococci and stated that

nature using the term *calymmatobacterium granulomatis*. Their work was confirmed by De Souza Araujo.

It is very doubtful whether the cultures obtained by all these observers are in reality cultures of Donovan's bodies. The inoculation of vaccines made from such cultures do not induce any improvement.

**Communicability**—The disease is generally transmitted by sexual intercourse.

**Pathology.**—According to Galloway, the microscopical changes begin some distance from the lesion, and consist of a round-celled infiltration into the upper regions of the corium. This induces

original length.

The connective tissue of the corium swells and disappears, and its place is taken by a round-celled infiltration, which consists of leucocytes, Unna's plasma cells, mast cells, and connective-tissue cells. Giant cells are not found. The leucocytes are the ordinary polymorphonuclear leucocytes while Unna's cells are characterized



FIG. 804.—GRANULOMA INGUINALE  
(From a photograph by Sambon.)

by possessing a rounded nucleus, with a certain amount of surrounding connective tissue. The connective tissue is shown showing mitosis. The stratum granulosum fails to develop its keratin hyalin granules, and eventually disappears halfway up the papule, as does the stratum corneum, so that on the summit the different layers of the epidermis cannot be differentiated.

There is neither caseation nor suppuration, but in the older parts of the specimen the cells of the infiltration become swollen and disappear, and in their place there appears cicatricial connective tissue, which causes the papule to shrink and the whole area to assume a scar-like appearance.

the scrotum and the thighs and from thence downwards into the perineum and around the anus into which it may pass

When fully developed it appears as a mass of nodules or papules without deep ulceration as a rule but with a thin offensive discharge. In the older regions it shows some attempt at healing in the formation of dense scar tissue. There is very little pain or pruritus.

In the female the process begins as a papule on the labrum minus and then extends into the vagina along the perineum around the



FIG 865 —GRANULOMA INGUINALE  
(From a photograph by Sambon)

anus and up the rectum and into the groins. The growth extends into the tissue between the rectum and the vagina and may give rise to rectovaginal fistulae. The whole growth is also much more liable to ulcerate in the female than in the male.

The lesions may become œdematous and present an appearance analogous to elephantiasis.

Bonne and Verhagen have described a case in which the disease after a time attacked the upper lip and the nose.

**Varieties**—Daniels lays stress upon the fact that the disease varies much in different races. In negroes it is more granular and spreads farther; in Indians it is less marked, in Fijians it is softer



absence of the secondary eruption and the inefficacy of mercurial treatment. Lupus is very rare in such situations and in epithelioma the lymphatic glands would be early infected. In doubtful cases microscopical examination of a piece of the diseased tissue will enable the diagnosis to be made from lupus as well as from epithelioma.

**Prognosis.**—The disease as a rule does not affect the general health, but is extremely chronic, lasting for years.

**Treatment.**—Tartar emetic, though not efficacious in every case, should always be given a thorough trial. The treatment is carried out in the same way as for cutaneous leishmaniasis (see p. 2173).

Greig and Curjel consider that tartar emetic benefits the condition only

### PAPILLOMA INGUINALE TROPICUM.

Syphilis is not a cause of this disease. The disease is not rare in the tropical regions, but is probably show that other races are also affected. Women are apparently more liable to the disease than men, inasmuch as although the number of male patients in the Colombo Clinic and hospitals is much larger than the number of female patients, not a single case has been detected among men.

The disease is very chronic, and is probably contagious, but nothing is known of the aetiology. In the cases so far observed the genital organs of the patient were normal and there was no history of gonorrhœa or any other venereal disease.

**Treatment.**—The treatment is surgical—removal of the vegetation by the knife and cauterization. The patient, as a rule, however, will not consent to the operation.

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## CHAPTER XCVI

# THE DERMATIZOIASSES

Classification—Hexapode dermatites—Creeping eruption—Circinate creeping eruption—Dermatitis macrogyrata—Chilopode dermatites—Acarine dermatites—Copra itch—Grain itch—Scabies—Nematode dermatites—Cestode dermatites—References

### CLASSIFICATION.

THE term dermatiziasis in the widest sense of the word means any skin disease of animal origin but it is usually restricted to indicate those skin lesions which are caused by metazoan parasites. Used in this restricted sense dermatiziasis includes —

- I Hexapode dermatites
- II Chilopode dermatites
- III Acarine dermatites
- IV Nematode dermatites
- V Cestode dermatites

### I. HEXAPODE DERMATITES

The *Hexapode Dermatites* include the lesions of the skin caused by either the bites, the stings, or the presence of the larvæ or the pregnant female of various species of the *Hexapoda*.

These hexapode dermatites may be divided into four classes —

- 1 Dermatitis caused by bites of the adult
- 2 Dermatitis caused by blistering fluids excreted by the adult
- 3 Dermatitis caused by stings of the adult
- 4 Dermatitis caused by the presence and bites of the larvæ
- 5 Dermatitis caused by the presence of the imago

#### ||1 Dermatitis caused by Bites of the Adult.

These lesions are most commonly due to bites of species of the families Pediculidæ (p. 753) Cimicoridæ (p. 762), Anthocoridæ (p. 774), and Culicidæ (p. 774).

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837)  
ants

The bites of these insects are either considered in pp 223-226 or in the references given above, and need not be further considered except with regard to the Pediculidæ which cause the dermatosis called Pediculosis.

### Pediculosis.

**Synonyms.**—Phthiriasis, Virgibond's disease

**Definition.**—Pediculosis is a term applied to the various lesions,

where it is an everyday sight to see the lower-class natives busily employed in killing the lice in their friends' heads. It is also commonly present in all armies on active service and as lice are carriers of such diseases as typhus relapsing fever and trench fever, etc., they have assumed a very important position in the recent war.

**Ætiology.**—Pediculosis is due to the irritation caused by the venom injected during the bites of the three species of lice mentioned in the definition.

**Pathology.**—The mouth parts of a louse consist of two tubes one inside the other, the outer chitinous tube called the *proboscis*,

upon the skin. It then protrudes the suctorial tube, which it drives deep into the skin of the host until it reaches the blood. It is during this process that it probably injects the venom from its salivary glands but the nature of this poison is quite unknown, altho

pumping the blood into its alimentary canal by means of the chitinous pharyngeal pump. After feeding it withdraws its proboscis and the blood fills up the orifice and coagulates, forming a minute red papule. Considerable pruritus is now felt, and the victim scratches vigorously to relieve this sensation and often produces marked excoriations which may become secondarily infected with the common pyogenic cocci causing purulent lesions. Repeated biting, associated with injection of the venom and constant scratching leads to pigmentation of the skin, causing

not be so entirely dependent upon the scratching as is usually  
 venom  
 pruritus which  
 accompanied by the  
 centres but this  
 simple picture is generally complicated by the erythema or ex-  
 coriations set up by the scratching induced by the pruritus and  
 this again may be complicated by the appearance of pustules  
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may be found on a fair European  
 the head and may give rise to secondary impetiginous lesions and  
 enlargement of the lymphatic glands especially those of the back  
 they may cause  
 polonica

Geer 1778 These pediculi live in the clothing especially in thick  
 seams and are therefore to be found on natives in the region of the  
 waist where the clothing is twisted into a thickish roll In Euro-  
 peans the most common site for their attacks is the back of the  
 shoulders where the small papules with the bright red centres and  
 the linear scratches may be seen

*Pediculosis pubis*—*Phthirus pubis* lives wherever there are  
 large thick hairs—viz on the hairs of the pubis or the eyelids  
 or eyebrows and of the beard and armpits Here again it is accom-  
 panied by the characteristic signs. In addition Morrison's spots  
 or maculæ ceruleæ may be seen in the form of small roundish or  
 oval greyish blue maculæ which are thought by some authorities  
 to arise from the pigment on the thorax of the louse opposite the  
 anterior pair of legs but which more probably arise from the action  
 of the venom The reddish deposits seen on the hairs are said to  
 The eggs may be seen as small oval

history of pruritus  
 some given region  
 with the little papules  
 the clothing which  
 the rare diffuse p g  
 for Addison's disease

or the suprarenal form of malaria, but may be recognized by the finding of the lice, the non diminution of the muscular power, and by the presence of the parasites. From scabies it may be dis-

hair little by little with carbolic acid (1 in 40) or soak long hair in

mable substance

Impetigo contagiosa may be treated by an ointment composed of ammoniated mercury (5 grains) and lard (1 ounce) and in children the hair may be cut and this ointment may be applied

*Pediculosis corporis*—The clothing and the bedding must be disinfected by steaming or boiling and the patient must have several large baths with free use of soap and water, as well as a soothing calamine lotion (40 grains calamine to 1 ounce of water) for application to the irritated skin. Lice destruction is more fully detailed on pp 1338 1339

It is important to remember that the eggs of *P. corporis* are often attached to the lanugo hairs. Merely cleaning the clothing is often useless. Rub or spray the whole body with paraffin and take a warm bath.

Martini recommends a depilatory consisting of strontium sulphate 2 parts zinc oxide 1 part talc 1 part. This is mixed with a little water and applied as a paste for ten minutes when it is removed and some olive oil used to soothe the irritation.

*Pediculosis pubis*—A white precipitate ointment (5 to 10 per cent) or an ointment of oleate of mercury (5 per cent, 6 drachms) with ether (2 drachms) will kill parasites and ova after which calamine lotion may be applied to allay the irritation.

#### Clinocerosis.

E 765

#### Siphonapteriasis.

Camp 101 may be used



## Formicidas

solution of carbolic acid (1 in 20) and as a preventative against ants infecting beds some powdered camphor may be dusted in the sheets (see p 222)

## 2 Dermatitis caused by Blistering Fluids excreted by the Adult,

The insects which act in this manner are the well known 'blister beetles' (vide p 226) which cause the eruption called Seasonal bullous dermatitis (synonym Seasonal vesicular dermatitis) which we will now describe

## SEASONAL BULLOUS DERMATITIS

**Definition**—Seasonal bullous dermatitis is characterized by an epidemic of bullæ of varying definite season of the year sensations of itching burning

**Historical**—Beetles have been known to cause blisters and

these insects and Linnæus Fabricius and Latreille gathered together quite an amount of information on these beetles and their varieties

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though doubtless many more insects can do so equally well —

*Staphylinidae* —

" "

*Cantharida* —*Epicaula sapphirina* Macklin 1845.*Epicaula tomentosa* Macklin 1845

blisters may extend a considerable distance along the forearm or down the back.

Usually there are no immediate symptoms and it is only after an interval of twelve to twenty four hours that an itching or burning sensation or even severe pain invites attention to the affected area when the blisters or blisters varying in size and number as already stated are to be found full of yellowish serum and situated on an erythematous areola.

As a rule the victim does not see the insect and may not remember one crawling on him and he may be entirely at a loss to account for the blisters. It is here that the difficulty of diagnosis arises in that the practitioner may see only one or two cases and at the moment may not think about these insects.

If pricked and carefully treated they quickly vanish and cause no further trouble but if they burst and are allowed to be rubbed by the clothing they become raw very tender and painful—a condition which may last for days.

More rarely a considerable portion of the blistering liquid appears to get well rubbed into one spot and then a small white eschar is formed which may be surrounded by an extensive inflammatory areola with its surface raised above the central necrotic area and covered with numerous small red papules. The whole region becomes very painful and tender and some couple of weeks elapse before healing is completed which generally takes place without any cicatrization. Secondary septic infections are rare. After being *en evidence* for some three to four weeks the beetles disappear and the epidemic ceases for the year.

**Diagnosis.**—The characteristic features of seasonal bullous dermatitis are as follows —

1 The sudden appearance of bullæ, varying in size and number, surrounded by a certain amount of inflammatory redness

2 The persons in whom the bullæ are found are usually in good health, and as a rule they are unable to assign a cause for

affected  
 ten in a row  
 portion of the body is

6 A number of healthy people living in the same place may be similarly affected at the same time.

season of the year  
 following

differentiated from the bullæ caused by burns and scalds, and by chemicals, by the history of the case.

2 It has also to be differentiated from the various forms of Hydroa as follows —

(a) It can be separated from the milder forms of dermatitis herpetiformis by the absence of severe itching and of circinate and papular erythematous lesions and by the absence of the tendency to be arranged like herpes

seasonal variety by only appearing in winter  
 festival in not being papulo vesicular in character  
 differentiated from herpes zoster by the absence of appearance of  
 in Head's areas  
 by the absence

dermatitis venenata,

presence of urticaria  
 6 It is easily separable from Dermatitis caused by insects as these give rise to small wheals and vesicles and not to bullæ

7 Ant and tick bites, stings of wasps bees scorpions, centipedes etc., are at once differentiated because the eruption in the present

differentiated clinically from  
 so severe as the eruption described by P. Darwin, in 1845

tion and cicatrization are absent but perhaps these may be only differences in details and not in essentials

**Prognosis**—This is good. Cases recover fairly rapidly and as a rule without cicatrization hence the outlook as regards rapidity of cure and the absence of scarring is good

It will however be remembered that P. Da Silva describes a much severer form of dermatitis than that mentioned here and that this was followed by cicatrization

**Treatment**—The best treatment is to prick the blister and apply a dressing of 1 in 80 carbolic acid but the majority of the victims just let the lesions alone and they heal up rather more slowly than when treated and are more painful. If they become rubbed they are often very painful

### 3. Dermatitis caused by Stings of Adults

Stings are mainly caused by species belonging to the family Apidae of the Hymenoptera which includes the bees and wasps. A description of these stings will be found on pp. 219-222

### 4. Dermatitis caused by Larvæ

See also p. 219 of the *Report on the Medical and Veterinary Services in the Sudan*

### BLOOD SUCKING DIPTEROUS LARVÆ

Only a very few blood sucking dipterous larvæ are known and these belong to two genera which may be distinguished from one another as follows—

- A. Abdomen long and narrow with unequal segments and distinctly longer than the thorax—*Auchmeromyia* Schiner and Bergenstamm 1819
- B. Abdomen short and broad with equal segments and but little longer than thorax—*Charomyia* Roubaud 1911

There are two species belonging to the last named genus—viz—

*Charomyia charophaga* Roubaud 1911

*Charomyia boneti* Roubaud 1911

Neither are known to attack man. They live in the burrows of the wart hog and the ant bear in the Sudan. There are also two species belonging to *Auchmeromyia*—viz—

*Auchmeromyia luteola* (Fabricius 1805)

*Auchmeromyia prægrandis* Austen 1910

Both these may attack man

**Diagnosis.**—The characteristic features of season 1 bullous dermatitis are as follows —

1 The sudden appearance of bullæ varying in size and number surrounded by a certain amount of inflammatory redness

2 The persons in whom the bullæ are found are usually in good health and as a rule they are unable to assign a cause for

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6 A number of healthy people living in the same place may be similarly affected at the same time

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the bullæ caused by burns and scalds a history of the case

2 It has also to be differentiated from the various forms of Hydroa as follows —

(a) It can be separated from the milder forms of dermatitis and of circinate and tendency

iformis called hydroa  
er size of its bullæ

which do not appear in

(c) From dermatitis recurrens it can be distinguished from the hermal variety by only appearing in warm weather and from the

5 It can be recognized as due to plants by the absence of the marked œdema and erythema which generally attack the face hands and genitalia and by the presence of bullæ

6 It is easily separable from Dermatitis caused by mites as these give rise to small wheals and vesicles and not to bullæ

7 Ant and tick bites stings of wasps bees scorpions centipedes etc are at once differentiated because the eruption in the present

## HEXAPODE DERMATOSES

tion and cicatrization are absent but perhaps these may differ in details and not in essentials

**Prognosis**—This is good. Cases recover fairly rapidly as a rule without cicatrization hence the outlook as regards cure and the absence of scarring is good. It will however be remembered that P. D. 1112 describes a severer form of dermatitis than that mentioned here and this was followed by cicatrization. **Treatment**—The best treatment is to prick the blister and a dressing of 1 in 80 carbolic acid but the majority of the cases just let the lesions alone and they heal up rather more slowly when treated and are more painful. If they become rubbed are often very painful.

### 3 Dermatitis caused by Stings of Adults

Stings are mainly caused by species belonging to the families of the Hymenoptera which includes the bees and wasps. A description of these stings will be found on pp. 219-22.

### 4 Dermatitis caused by Larvæ

The larvæ of various species of the (Estridæ and Muscidæ) are compelled to undergo their development in the skin of some warm-blooded animal and as these are plentiful as a rule the flies do attack man who only occasionally suffers from their effects when he does the pathological condition is usually named dermatomyiasis (p. 163). Other larvæ—as for example that of *Auchmeromyia luteola* Fabricius 1805—are blood suckers.

### BLOOD SUCKING DIPTEROUS LARVÆ

Only a very few blood sucking dipterous larvæ are known and these belong to two genera which may be distinguished from one another as follows—

- A. Abdomen long and narrow with unequal segments and distinctly longer than the thorax—*Auchmeromyia* Schiner and Bergenstamm 1819
- B. Abdomen short and broad with equal segments and but little longer than thorax—*Cheromyia* Roubaud 1911

There are two species belonging to the last named genus—viz—  
*Cheromyia cherophaga* Roubaud 1911  
*Cheromyia boneti* Roubaud 1911  
They live in the burrows of the wart hog and the ant bear in the Sudan. There are also two species belonging to *Auchmeromyia*—viz—  
*Auchmeromyia luteola* (Fabricius) 1805  
*Auchmeromyia* sp.



**History**—This disease was first described by A Lee in 1875. Later on Procke, Blanchard, Topsent, Fulleborn, Macfie and others have recorded several cases. It is not rare in some parts of Europe, Africa and Asia and in South America. We have seen numerous cases in Ceylon. It is extremely rare in North America.

**Ætiology and Pathology**—Larvæ of the genera *Gastrophilus*, *G. hæmorrhoidalis* and *G. nasalis*, *Æstrosia satyrus*, *Hypoderma*



FIG. 866.—LARVA MIGRANS

*bovis* and *H. lineata* have been found in several cases. In others no larva whatever was found. Looss states that the same clinical picture may be caused occasionally by ancylostoma and strongyloides (*Ancyllostoma*) larvæ or even by an inanimate object like a piece of horsehair.

**Symptomatology.**—The eruption is characterized by the presence of a narrow raised red line  $\frac{1}{8}$  to 1 inch broad. This line extends daily one or several inches and is generally sinuous but may be



straight While the advancing end progresses the opposite end slowly fades away The duration of the malady is long—generally several months but occasionally two or three years There is much pruritus

**Treatment**—Hypodermic injections of various disinfectants have been tried with little success Hutchins recommends a cocaine injection followed by the injection of 1 or 2 drops of chloroform

### Circinate Creeping Disease.

In Ceylon cases are met with of a peculiar eruption which is possibly of the same nature as the creeping disease previously described though larvæ were not found

The condition is characterized by the presence on the back of the hands of a ringed eruption with markedly elevated thick angry red borders. I after the patient has may be one ring or s

The rings expand excentrically The patients complain of the extreme irritation and in all the cases stated that they felt some thing creeping along the red circles as though a worm had got inside in handling the turf The duration varies but it generally does not exceed two or three weeks

search for larvæ has been fruitless is present

**Treatment**—Liq plumbi and other soothing applications are generally useless The best results are obtained by continuously applying on lint a diluted alcoholic solution of resorcin —

Resorcin  
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### Dermatitis Macrogyrata

dermis The eruption is associated with pain sometimes severe but there is seldom any pruritus The condition is most persistent, and in our cases neither fly larvæ could be found nor fungi isolated In none of our cases was there history of syphilis, and potassium iodide and mercury had no effect The treatment is most unsatisfactory Antimycotic substances such as chrysarobin and tincture of iodine have no effect The application of a lotion of liquor

plumbi (3ii), tincture of opium (3i) diluted with 8 ounces of water, or of dressing soaked in 0.5 per cent of resorcin may cause a slight improvement



FIG. 867.—*DERMATOPHILIASIS MACROGYRATA*

##### 5 Dermatitis caused by the Presence of the Imago.

At times the impregnated female insect burrows into the skin while the eggs mature

A good example of this is *Dermatophilus penetrans* Guérin 1838

##### Dermatophiliasis (Jigger).

**Synonyms**—Nigua (Honduras), Chique (Salvador) Chica (Columbia), Bicho Tunga (Brazil), Pique (Argentine) Chique (French Colonies)

**Definition**—Dermatophiliasis is the invasion of the skin and subcutaneous tissue by the pregnant female jigger (*Dermatophilus penetrans*)

**Remarks.**—The home of the jigger is in tropical America from 23° N to 28° S but it and its wanderings over the world have been sufficiently described on p 862 At present it is found in South America West and East Africa Madagascar Uganda India, and, it is said China

**Symptomatology.**—The symptoms begin with itching and irritation generally in some part of the foot especially the toes and often under the toe nail. On inspection a small dark dot (the last) is seen in the skin. If the parasite develops around the dot it gives rise to a swelling which may attain the size of a small pea in the centre of which is seen a depression containing the black dot and finally ulceration takes place and the body of the parasite is thrown off but not until all the eggs are laid.



FIG 868.—DERMATOPHILIASIS (After Newstead)

- a Group of jiggers b isolated jiggers c pits left by jiggers  
d lateral view of jiggers deep in the skin

When the parasite has been dislodged by treatment or suppuration a small ulcer is left which is very liable to septic or other bacterial infection.

In cases of heavy infection Quérois recommends the use of petroleum or of an ointment consisting of salicylic acid 1 part ichthyol 4 parts vaselin 4 parts

**Prophylaxis**—Prophylaxis consists in keeping the house clean and pigs poultry and cattle kept away therefrom High boots should be used and especial care should be taken not to go to a ground floor bathroom with bare feet The feet especially the

Jeyes fluid or with pyrethrum powder or with a strong infusion of native tobacco as recommended by Low and Castellani

## II CHILOPODE DERMATITES

## III ACARINE DERMATITES

The *Acarine Dermatoses* include the skin lesions caused by the ticks and mites The tick bites are described on pp 215 and 217 The mites (pp 690 693 and 724 732) which most commonly attack man are —

### DERMANYSSINÆ

*Dermanyssus gallinæ* de Geer 1778

*Dermanyssus hirudinis* Hermann 1804 — These mites produce a papular eczematous dermatitis in poultrymen

*Holothyrus coccinea* Gervais 1842 cause a swelling in the part attacked

### TROMBIDIDÆ

*Microtrombidium akamushi* Brumpt 1910 is the cause of Tsutsugamushi disease

*Microtrombidium holosericeum* Linnæus 1746 has a larva (*Leptus aut in* the autumn effective pres used — Oil of lavender ʘʘʘ spirit of camphor ʘʘʘʘ

'pou dagonte of Guana the maibi of New Granada the 'colorado of Cuba the mouqui of Para and the bete rouge of Martinique and Honduras are not known  
*Trombidium wichmanni* Oudemans 1905 is the gonone of

in North Queensland

#### TETRANYCHIDÆ

*Tetranychus molestissimus* Weyenbergh 1886 causes severe itching in the Argentine and Uruguay during the months December to February by thrusting its hypostome into the skin and thus causes 'Bicho colorado itch

#### EUPOPIDÆ

*Tydeus molestus* Momez 1889 is the cause of Guano itch

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 the worm of cotton is  
 st and from this  
 infected cotton in

re also known to

attack man

#### TYROGLYPHIDÆ

*Tyroglyphus longior* Gervais var *castellanus* Hirst 1912 is the cause of copra itch

*Tyroglyphus siro* Linnæus 1758 and *Aleurobis farinae* de Geer are believed to be the cause of vanillism

*Glyciphagus prunorum* Hermann is the cause of grocer's itch

*Rhizoglyphus parasiticus* Dalgetty 1901 is the cause of a type of so called coolie itch of the feet Bell states that it causes a large circular superficial sore on the sole of the foot This is produced by numbers of the parasite invading the skin

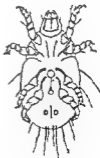


FIG 869—*Rhizoglyphus parasiticus* MALE

(After Dalgetty)

#### SARCOPTIDÆ

*Sarcoptes scabiei* var *hominis* Linnæus 1758 is the cause of scabies in man

In addition to this common parasite there are several varieties usually occurring in the domestic animals which may at times attack man—e.g. *Sarcoptes scabiei* var *canis* found in the dog *S. scabiei* var *ovis* in the sheep

*S scabies* var *equi* in the horse, *S scabies* var *simi* in the pig *S scabies* var *auchenica* in the lama *S scabies* var *cameli* in the camel.

*Notadres cati* var *cati* Hering 1838 found in cats may occur in man.

#### DEMODICIDÆ

*Demodex folliculorum* Simon 1842 is said to be the cause of certain inflammations in seborrhœa. Some authors consider them to be of importance in the carriage of certain diseases such as leprosy and cancer.

Rarer *Acarinae* which attack man occasionally are *Trombidium striaticeps* Heim and Oudemans 1904 on fowls and dogs *T. americanum* Riley and *T. irritans*. *Metatrombidium poriceps* Heim and Oudemans 1904 on fowls and dogs. *Urotrombidium merrilli* Hale and *M. pusillum* Hermann.

#### Copra Itch

**Definition**—A very pruriginous dermatitis found in people handling copra and caused by *Tyroglyphus longior* Gervais var *castellani* Hirst (p. 729).

**Historical and Geographical**—This dermatitis was described by Castellani in 1911 who observed it in Ceylon in people handling copra and considered it to be due to an acarus like parasite swarming in many samples of copra. He sent the specimens of the parasite to Hirst who described it as a new variety of *Tyroglyphus*—*Tyroglyphus longior* Gervais var *castellani* Hirst.

Castellani Hirst recently confirmed by Graham

huge numbers in certain samples of copra and may occasionally be found on the skin of the patients but remains on the skin only temporarily as it does not burrow itself. It apparently induces

the dermatitis in the same manner as *Pediculoides ventricosus* Newport which lives in diseased cereals produces an eruption in



FIG. 870. MICROPHOTOGRAPH OF THE TYROGLYPHUS OF COPRA ITCH (X40)

pruriginous urticarial or papuloid eruption often develops. The same result is obtained by picking the mites out of copra dust and

'pou d'agonte' of Guiana, the 'nabi' of New Granada, the 'colorado' of Cuba, the 'mouqui' of Para, and the 'bête rouge' of Martinique and Honduras, are not known

*Trombidium wichmanni* Oudemans, 1905, is the gonone of Celebes whose larvæ attack man, burrowing into the skin as just described above

*Trombidium nann* Oudemans 1905 is the gonone of  
*rombidium* is reported  
 in North Queensland

#### TETRANYCHIDÆ

*Tetranychus molestissimus* Weyenbergh, 1886 causes severe itching in the Argentine and Uruguay during the months December to February by thrusting its hypostome into the skin and thus causes 'Bicho colorado itch'

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#### TYROGLYPHIDÆ

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*Glyciphagus prunorum* Hermann is the cause of 'grocer's itch'

*Rhizoglyphus parasiticus* Dalgetty, 1907, is the cause of a type of so-called 'coolie itch' of the feet. Bell states that it causes a large circular superficial sore on the sole of the foot. This is produced by numbers of the parasite invading the skin



FIG. 365.—*Rhizoglyphus parasiticus*  
 MALE

(After Dalgetty)

#### SARCOPTIDÆ.

*Sarcoptes scabiei* var *hominis* Linnæus, 1758 is the cause of scabies in man

In addition to this common parasite there are several varieties, usually occurring in the domestic animals, which may at times attack man—e.g. *Sarcoptes scabiei* var *canis*, found in the dog, *S. scabiei* var. *ovis* in the sheep,

*S. scabies* var *equi* in the horse, *S. scabies* var *suis* in the pig, *S. scabies* var *auchenia* in the lama, *S. scabies* var *cameli* in the camel.

*Notedres cati* var *cati* Hering 1838 found in cats may occur in man.

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**Historical and Geographical**—This dermatitis was described by Castellani in 1911 who observed it in Ceylon in people handling copra and considered it to be due to an acarid like parasite swarming in many samples of copra. He sent the specimens of the parasite to Hirst who described it as a new variety of *Tyroglyphus*—*Tyroglyphus longior* Gervais var *castellani* Hirst.

Castellani's researches have been recently confirmed by Graham Little, Whitfield and Ditlevsen.

**Ætiology**—The mite is present in huge numbers in certain samples of copra and may occasionally be found on the skin of the patients but remains on the skin only temporarily as it does not bury itself.



FIG. 870.—MICROPHOTOGRAPH OF THE TYROGLYPHUS OF COPRA ITCH (X 40)



mite or the copra dust containing it

**Symptomatology**—The hands arms legs and sometimes the whole body except the face present fairly numerous very pruriginous papules often covered by small bloody crusts due to scratching papulo pustules and pustules are also generally present The eruption has no tendency to spontaneous cure while the patient goes on working in the infected mills

**Diagnosis**—On superficial examination the condition may be easily mistaken for scabies but burrows are not present and the two parasites are very different

**Treatment**—The best treatment is the daily application of  $\beta$  naphthol ointment (5 to 10 per cent) The action in these cases cannot be compared to what takes place in scabies because in copra itch the acarus like parasite remains for only a short time on the body and in most cases when the ointment is applied at night the mites are no longer there It may act as an antipruritic antiseptic and in this way diminish scratching and secondary pyogenic infections It is probable also that a small amount of the ointment may remain on the skin after the morning bath and be repellent to the mite in this way preventing the daily reinfection which otherwise takes place

### Grain Itch

**Synonyms**—Straw itch Barley itch Dermatitis Schambergi Urticarioid Dermatitis Dermatitis Distropenotus Aureoveridis Acara Dermatitis urticarioides Schamberg's disease Straw mattress disease Cotton seed Dermatitis

**Definition**—Grain itch is a dermatosis caused by *Pediculoides ventricosus* Newport 1850 (*vide* Figs 343 345 p 728)

**History**—During the last fifty to sixty years this disease has been recognized in Europe but only since 1901 in America when Schamberg described it In 1909 Goldberger and Schamberg found that it was caused by the same mite as in Europe Since 1914 when

*grande* Riley) the joint worm (*Issoma tritici* Hitch) the Angoumois grain moth (*Sitotropa cerealella*) and the caterpillars of the

cotton moth (*Gelechia gossypiella*) The mite attacks people it  
 A prob  
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vesicles may at times pustules in a few hours Develt (dbs) o =

ing  
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 the  
 of the presence of this mite and search for it in grain or straw or cotton seed

Treatment—The treatment consists in removal of the cause—  
 of handling the infected grain—and the application of soothing  
 lotions such as calamine lotion or a dilute carbolic acid or acetic  
 acid lotion

### Scabies

Synonyms—Scabrities Psora (term wrongly applied) Itch,  
 Courap (=itch Bontius) Scabies indica (Savages) La gale  
 (French) Kraetze (German) Sarna (Madera) Scabbia (Italian)

Definition—Scabies is an infection of the superficial layers of the

itching Secondary lesions are vesicles (at which ticks o JF  
 is at the far end of the burrow immediately beyond which lies the  
 acarus) scratches scabs pustules and a superficial dermatitis

## V. CESTODE DERMATOSES

The cestode dermatoses are usually due to *Sparganum prolifer* Ijima 1905 (p 606) which produces nodules in the skin which are associated with considerable swelling thus giving rise to an appearance not unlike elephantiasis. In addition there may be an acne like eruption all over the body which is very irritable and causes pruritus. On scratching the papules and producing excoriations the worms may escape while on incising a nodule a cyst with one or two worms embedded in slimy jelly or a watery fluid may be found.

After lasting for some weeks or months the cyst walls become firm and thick and so encapsulate the worms. The condition may last for years. There is no known treatment.

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burg

## Acarine Dermatoses

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## Noxious Larvæ

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WHITE (1887) *Dermatitis Venenata* Boston

## Blood sucking Dipterous Larvæ

- DUTTON TODD AND CHRISTY (1904) *Liverpool School of Tropical Medicine*  
Vol 49 54 Liverpool  
GRAHAM SMITH (1914) *Non Blood Sucking Flies in Relation to Disease*  
Cambridge  
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551 554 Paris (1913) *Bulletin de la Société Pathologie Exotique* vi  
128 130 and *Bulletin Scientifique de la France et de la Belgique* xiv  
105 202

## CHAPTER XCVII

# DYSIDROSES AND DYSTROPHIES

Hyperidrosis Prurigo Dermatitis Eczema etc. Pruritus Prickly heat—  
 Xanthoderma areatum—Mongolian spots—Tattooing—Anthem—Sym-  
 metrical palmar erythema—Acrodermatitis vesiculosa—References

### HYPERIDROSIS.

Remarks.—This condition as well as bromidrosis, is a cosmopolitan

condition, occurring in all climates, and is often associated with pruritus, eczema, and other skin diseases. It is most common in the hot season, and is often associated with pruritus, eczema, and other skin diseases. It is most common in the hot season, and is often associated with pruritus, eczema, and other skin diseases.

the condition disappearing when the hot season is over, but it is often associated with pruritus, eczema, and other skin diseases.

Treatment.—For the general hyperidrosis common during the hot season we are not in favour of any drastic internal treatment such as the administration of belladonna. In fact, we think it may be dangerous to stop suddenly this hyperidrosis which is in reality merely a physiological fact. For such cases we simply recommend using some potassium permanganate, or cyllin, or a little menthol alcoholic solution in the daily bath, and dusting of the

less Sulphur is occasionally given by us in cachets (3 grains)  
varies according to the

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(1 p  
are very useful if there are excoriations or irritations of the skin  
no alcoholic lotions should be used but merely water solutions of boric  
acid (2 per cent) carbolic (1 per cent) permanganate of potassium (1  
in 4000) and occasionally hydrogen perchloride (1 in 2000 to 1 in 4000)  
after which a salicylic or boric powder is applied. It should be always  
remembered to sprinkle with the same powder the socks shoes and  
undergarments

### BROMIDROSIS

This term is used to denote offensive sweating

**Ætiology**—The bad odour seems to be due to the growth of various  
bacteria as observed by Thin and is due not only to the sweat but also  
and probably in a higher degree to the sebaceous secretion. The condition  
is very common in native races—negroes Indians and Chinamen most  
natives seem to have it to some extent in fact. On the other hand  
however it is to be noted that certain natives state that they can  
detect in almost every European a special disagreeable odour. Certain  
authorities are of opinion

In contrast to bromidrosis cases have been described of certain individuals  
having a pleasing smelling sweat with the odour of violets or musk. There  
is a tradition that certain saints exhaled a pleasant odour

**Prognosis**—Except in those cases when the bad smell is due to  
accumulated dirt—when a thorough washing with carbolic soap will  
cure the condition—bromidrosis is not of easy cure but the bad  
smell may be hidden in various ways

**Treatment**—This is the same as for hyperidrosis but formalin  
lotions (½ to 3 per cent) alcoholic or watery are especially useful.  
Lysolorm (2 to 5 per cent) is efficacious. Afterwards a powder  
such as ac salicyl gr x talci ʒi or ac borici ʒi talci ʒi should  
be used and some boric acid should be sprinkled in the socks and

also in the boots. If there are excoriations formalin should not  
 be used. *Urotropin* gr ʒi of  
 three times daily may be tried. One of us had good results in a  
 case by the administration of *urotropin* gr ʒi thrice daily.

#### Chromidrosis

The term is applied to coloured excretion of sweat or sebum. The condition affects in most cases the armpits but cases have been described affecting the face, chest, abdomen, inguinal regions, hands and feet. The colour has been described as black, blue, red, green, yellow and violet. We have personally observed only two cases of chromidrosis. In both the axillary regions were affected, the colour was brick red and the sweat stained the clothes red. In one of the cases it was due to *B. prodigiosus* in the other to a red pigment producing coccus.

#### Phosphoridrosis.

**Synonym**—Phosphorescent sweat.

This condition has been described by Koster and others but is very rare. In one case it was stated that it appeared after eating phosphorescent fish. According to Beyerink the phosphorescence is due to photo bacteria.

#### Uridrosis.

Small white crystals forming a sort of hoar frost are present on the skin, due to excretion by the skin of urinary constituents especially urea and chlorides. Nash records several cases of a whitish deposit on the skin in native children and natives which according to some authorities may have been the same condition.

#### Hæmatidrosis

Several cases of hyperidrosis with red blood cells and leucocytes in the sweat have been placed on record.

#### Anidrosis

Idiopathic total anidrosis or absence of sweat is exceedingly rare but a diminution in the secretion of sweat is often observed. There are people in whom the bringing about of perspiration by hot air baths and drugs is very difficult.

*Symptomatic anidrosis* is present in leprotic patches and may be of diagnostic value. It is seen also in scleroderma general or circumscribed (morphæa) and in xeroderma. The secretion of sweat may be much decreased in diabetes and certain nervous conditions.

### DYSIDROSES.

#### Prickly Heat.

**Synonyms.**—Lichen Tropicus, Sudamina, Papulosa, Miliaria Rubra, Miliaria Papulosa, Salpullido (Cuba), Calor Picante (Minorca), Humon El Nil (Arabic).

**Definition.**—Prickly heat is a papular or papulo-vesicular eruption with marked pruritus and associated with profuse sweating.

**Geographical Distribution.**—The condition is found all over the tropics and subtropics. It may be observed also in temperate zones during the hot season especially at sea bathing places.

History—Bontius described the affection in his work *De Medicina Indorum*. Cleghorn in his book *Diseases of Minorca*

complete paper on the condition he uses the term *salpullido*. In more recent times the eruption has been studied by Robinson, Torok, Durham and many others.

Ætiology and Pathology—Poltzer considers the disease to be due to the obstruction of the flow of sweat brought about by the cells

always about the sweat pore.

Durham believes prickly heat to be an infective disease caused by a minute actively motile amoeba; his results, however, have not been confirmed.

occasionally become purulent. Besides the papules, roseola-like spots are often seen; these in some cases may coalesce and form large erythematous patches. Occasionally minute glass-like vesicles of *sudamina crystallina* are also present. The eruption is found on the parts of the body where the patient perspires most. It is very commonly observed round the waist, the back, chest, arms, and forehead; it may extend to the whole surface of the body except it is

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by

the absence of any moist lesions during the whole course of the malady. Our experience is, however, that in some cases true eczema—especially of the papular type—develops on prickly heat lesions. In cases of generalized prickly heat, with roseola-like



spots on the palms and soles, acute patchy congestion in the oral mucosa and pharynx a syphilitic must be excluded for several of our patients believed themselves to be affected with syphilis, but the extreme pruritus is generally sufficient to exclude it

**Prognosis.**—As a rule, the prognosis is good, the eruption disappearing quickly under proper treatment. The patient, however, complains of the severe itching which often keeps him awake at night. In some few cases no treatment is of any avail and the patient must be sent up to the hills, in others, crops of boils develop, or pyosis mansonii or impetigo contagiosa may supervene, especially in children.

**Treatment.**—The patient must be kept cool, he should not take much to drink and should abstain from drinking hot tea. Too warm clothing should be avoided, as it makes prickly heat worse. Recommended but not in any violent exercise. Sea-bathing

which in our experience has answered best is the free use several times daily, of a salicylic alcoholic lotion (ac salicyl  $\mathfrak{z}$ i spir rect  $\mathfrak{z}$ viii), followed by the general application of a salicylic or boracic or camphor powder such as ac salicyl gr x talci  $\mathfrak{z}$ i, or ac boraci  $\mathfrak{z}$ i, talci ven  $\mathfrak{z}$ i, or camphor gr xxxv, zinci ox amyli aā  $\mathfrak{z}$ ss. It is better, a

After some cold water should be directed to use afterwards to apply one of the powders mentioned above ant in the bath, and

The so called Castellani's lotion much used in the East consists of menthol gr x ac salicyl gr ii zinci ox  $\mathfrak{z}$ vi calaminæ  $\mathfrak{z}$ iii spirit rect  $\mathfrak{z}$ ii glycerin  $\mathfrak{z}$ i aq rosæ ad  $\mathfrak{z}$ vi. It should be diluted with the same amount of water when applied to the face or when used for children.

### Chelropompholyx.

and is also met with in tem  
Tilbury Fox and J Hutchin

i. :

Unna Norman Walker Williams and others show that the vesicles are of an inflammatory character, the vesicles are found in the prickly layer, and often press to one side of the sweat channel.

Unna described a bacillus as the cause of the disease. Some authorities consider the condition to be of neurotic origin.

**Symptomatology.**—The eruption is found in individuals who suffer from hyperidrosis. It is characterized by the presence of deeply seated translucent or opalescent, sago-like vesicles between the fingers and toes, the vesicles are not, as a rule surrounded by

an inflammatory halo, they very rarely coalesce, and usually do not break, but dry up gradually, being thrown off with the exfoliating epidermis. The vesicles in many cases are few in number, in others

from eczema, the principal characters of differentiation being the deep situation of the vesicles, the fact that they very rarely rupture, and that, as a rule, they are not surrounded by an inflammatory zone, moreover, the parts affected are generally bathed in sweat

**Prognosis.**—The disease is not a serious one and, under proper

lotion, to which a little menthol and alcohol has been added, answers well, or a salicylic alcoholic lotion (ac salicyl ℥i, alcohol rect ℥iv., aq ad ℥viii) may be used

#### Dysidrosis exfoliativa.

This affection described by Castellani, is closely related to

of calamine lotion is useful

#### Sweat Desquamation.

Schorfberg has applied the term 'sweat desquamation' to the minute

### DYSTROPHIES.

#### Leucoderma.

**Synonyms**—Leucopathia Vitiligo Acquired leucopathia, Acquired leukasmus, Acquired achromia, Acquired piebald skin Maladie de dépigmentation (O'Zoux).

Leucoderma is much more common in tropical countries than in temperate zones. The natives of Ceylon and India have a dread of developing leucoderma and look upon it as a loathsome disease.



FIG 871 —LEUCODERMA IN A SINHALESE WOMAN

#### **Etiology and Pathology —**

The etiology is unknown. It is generally considered to be a trophoneurosis. Occasionally there is evidence of hereditary influences several members of the same family being affected. The malady may start without any apparent cause or in some cases may begin after an injury after a burn after too strong a caustication. In fact the application of strong remedies should be used with care in natives. We have seen leucoderma patches appearing after the application of pure formalin. Patches of leucoderma may develop also in chronic epiphytic skin diseases the fungi apparently having a deeply disturbing effect on the pigment formation.

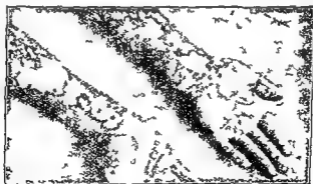


FIG 87 —LEUCODERMA OF THE HANDS AND ARMS

**Symptomatology —**Leucoderma is characterized by the presence of non pigmented areas white ivory like or pinkish. The patches are roundish or oval with a smooth surface they slowly enlarge and coalesce giving rise to large irregularly outlined areas. Occasionally almost the whole body becomes affected more often it is

the face and hands which are affected and there may be a certain symmetry. The initial patches are often surrounded by a zone of hyperpigmentation. Occasionally within the white areas small dot like zones of pigmentation are left. The hairs of the affected parts frequently become white but sometimes remain of normal colour. The white patches do not show any marked change in sensation there is never anæsthesia in many cases there is hyperæsthesia to heat and light stimulation. The texture of the skin remains normal occasionally slight atrophic processes may be noted.

It is usually stated that the general health is not impaired. In our experience however when the patches are large and situated on uncovered parts of the body especially the face symptoms of severe anaemia have been noticed. Moreover the patients complain that they cannot do any work in the sun as they experience a burning sensation in the white patches and they suffer from giddiness.

#### Clinical Varieties—

*Melung (Beta)*—This type of leucoderma was first described by Ziemann and is fairly common among West African negroes. It is found also in Ceylon, India and Burma in the last mentioned country having been described by Castor. The affection is always symmetric and attacks only the palms or the soles or both palms and soles. Small areas of the skin



FIG 873—LEUCODERMA IN A SINHALESE

undergo a slow process of depigmentation becoming whitish or yellowish there is no alteration of sensibility. The depigmented areas which are generally of various shape are intermixed with patches of normally pigmented skin so that the palms and soles present a marmoriform appearance.

The disease is chronic and incurable. It often develops in childhood and several members of the same family may be affected.

A variety of leucoderma in the shape of two small often triangular

has been observed by Pusey  
Ceylon The mucosa of the

skin; from tinea flava, tinea alba, and pinta, by the absence of any fungus.

**Prognosis.**—The disease may be said to be incurable. When large patches are present, the patient complains often of asthenia, and may become unfit for work, especially work in the open air and sun.

**Treatment.**—The disease is generally incurable, but the spreading of the patches may be prevented, and occasionally a slight improvement may be brought about, in our experience, by an energetic arsenical treatment. We generally give arsenious acid in a pill (gr  $\frac{1}{10}$ ) three to six times a day, or atoxyl injections (5 grains every other day). Gillmore has tried soamin with fairly good results. We have seen no benefit from the administration of suprarenal extract, as recommended by several authors. The white colour may be partially hidden by applying a lotion of nitrate of silver or potassium permanganate, or by tattooing.

Heidingsfeldt has devised an instrument consisting of a group of ten needles which are put in movement by electrical power. In this way tattooing may be performed much more rapidly than by hand.

Sommer claims to have cured several cases of leucoderma by injections of adrenalin.

### Albinism.

**Synonyms.**—Congenital leucoderma, Congenital leukopathia, Congenital achromia, Congenital leukasmus.

The affection is found in the tropics more frequently than in temperate zones. It is characterized by congenital absence of pigment in the skin, hair, iris, and choroid. There are cases, however, of partial albinism in which only the skin is affected. The skin has a milky white or pinkish appearance, the iris is rose-coloured, and the pupil red. There is intolerance to light; hence

feeble individuals.

**Ætiology and Pathology.**—The ætiology is unknown. The affec-

### Erythema Solare

The effects of sunlight on the skin including the histology of the lesions have already been discussed in Chapter III pp 83-85. They are caused by the active effects of the rays at the violet end of the spectrum. The skin of the parts exposed becomes erythematous; swollen and vesicles and bullæ may appear. Desquamation follows and the skin is often left pigmented (sunburn).

The treatment consists in applying calamine lotion and later any bland ointment such as simple cold cream.

### Dermatitis Solaris

After repeated attacks of erythema solare—or at times without any history of such—the skin of the hands and exposed parts in planters and other people living an outdoor life in the tropics becomes slowly reddened and may be slightly rough to the touch. Freckles and hyperpigmented spots are generally present and not rarely small telangiectasia. In a later stage warty patches often appear and the dermatitis as noted by McLeod may somewhat resemble the dermatitis produced by X rays. Atrophic changes may also develop. The condition, which is also known by the term *tropical skin* is somewhat similar to what Unna called *seaman's skin* and to senile atrophodermia or biotripsy (see p 2282).

**Diagnosis**—The diagnosis from pellagra has already been discussed (see p 1730).

**Prognosis**—The dermatitis is very obstinate but generally becomes cured spontaneously in a cold climate.

**Treatment**—A change to a temperate climate is the only efficacious treatment. Exposure to the sun is to be avoided as much as possible.

### Chloasma

Chloasma which as is well known is characterized by the presence of dark brownish or dirty yellowish patches situated commonly on the face of a woman, is a condition that occurs in tropical climates as well as in temperate zones. It is a condition of the skin that is caused by exposure to the sun. They are seen especially on the face of a woman.

A similar condition may be due to exposure to a powerful

to a deep black bronzine one (see *Chloasma Bronzinum*) On close

*erythema solare* in one of our cases they appeared on the forehead twenty four hours after a motor car drive in the midday sun without the hood on in another a European lady who had a very delicate skin and was used to wearing gloves very dark hyperpigmented patches appeared on the back of the hands and wrists twelve hours after exposing her hands without gloves to the midday sun In addition to hyperpigmented spots depigmented patches also developed

2 *Chloasma calorificum*, from exposure to heat or possibly the glare of fires We have seen it several times in stokers

3 *Chloasma traumaticum*, from mechanical irritation of any kind scratching etc

4 *Chloasma toxicum*, due to irritating drugs as for instance after a blister

type of *chloasma symptomaticum* is found in patients suffering from chronic malaria and kala azar A diffuse type of hyperpigmentation observed in chronic malaria and closely resembling Addison's disease has already been described (see p 1180)

Hyperpigmentation may occur also in syphilis leprosy tuberculosis diabetes and many other chronic diseases

In India a pigmentary fever has been described of short duration and said to be characterized by the appearance of hyperpigmented patches on the face (see p 1461)

### **Chloasma Symmetricum**

This condition which has been described *Castellani* is often met with in Sinhalese who greatly object to it It is characterized by the presence of two dark brownish *chloasma* patches situated symmetrically one on each cheek generally on the malar region In some cases in addition to these two patches a third one is found on the nose The colour of the patches is generally dark brownish very rarely bronzine The causation is unknown it does not seem to be congenital No treatment is of any avail

### **Chloasma Bronzinum**

This somewhat rare affection is met with among natives as well as Europeans in India Ceylon the Malay States China and other

PLATE XIV



CHLOASMA ET AL.







**Dermatosis Festonata Frontalis**

**Historical and Geographical**—This condition has been described by Castellani in Ceylon. It has recently been found also in Macedonia.

**Ætiology**—This is unknown but probably a continuous exposure to the sun and glare plays a certain role in the causation of the malady.

**Symptomatology**—The affection which in its severe type is rare is found in Europeans who have resided for many years in the tropics and who have lived an open air life such as planters and overseers

argin  
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hitish

occasionally leucoderma like appearance and may be slightly

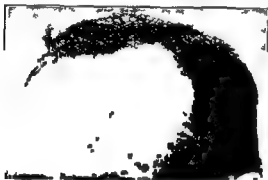


FIG 875—DERMATOSIS FESTONATA FRONTALIS

atrophied at times small patches of hyperpigmentation may be present. There is very little or no pruritus and sensation to the

slowly expanding  
tendency to spon

spontaneous cure

**Diagnosis**—The affection is not rarely taken for a trichophytic condition but the microscopical examination for fungi is always

The affection runs an extremely long course and has no tendency to spontaneous cure in the tropics. It gets much better if the patient goes to reside in a cold country.

**Treatment**—The treatment is very unsatisfactory. The patient must be advised not to expose himself to the sun and glare though it is doubtful whether the condition is directly due to such exposure. A wide brimmed topee or sun helmet lined with red cloth may be used.

Of the many drugs tried ichthyol seems to be the only one which at times keeps in check the condition occasionally inducing a slight improvement. It is given internally in 5 grain doses three times daily before meals and an ichthyol ointment or lotion (5 per cent) may be applied to the affected skin at night while during the day a calamine lotion may be used.

Mercury potassium iodide and arsenical preparations are useless.

#### Dermatosis Nigro-Anulata

**Historical and Geographical**—This condition has been described by Castellani in Ceylon natives and in a very dark skinned gypsy in Macedonia.

**Etiology and Pathology**—This is unknown. It is not a frambœside as the lesions are not influenced by salvarsan and potassium

encircling apparently normal skin. There is no pruritus sensation normal Wassermann negative. The course is extremely long lasting for years with very little change in the aspect of the lesions.



FIG. 876.—DERMATOSIS NIGRO-ANULATA

**Diagnosis**—The absence of pruritus, the multiplicity of the lesions, the absence of a mycotic or potyvirus nature of the lesions.

**Prognosis**—The general health is not affected but the condition is most persistent.

**Treatment**—This is very unsatisfactory. An exfoliative treatment by means of a salicylic acid ointment occasionally induces a temporary improvement.

### Ochrodermatosis

**Synonym** — The yellow disease (Castellani)

**Historical and Geographical** — This condition has been described by Castellani in Ceylon in Europeans living in the low country.

**Ætiology** — Unknown. The patients were not taking any drug and were not exposing themselves to the action of any toxic substance. On the strength that the condition gets much better and disappears on the patient going to the hills, a search for a possible

the sweat is not coloured, the stools are normally pigmented, the liver and spleen are not enlarged, and the general health is in no way affected, but naturally the patients greatly object to the disfigurement. The condition improves or disappears on the patient going to the hills or to Europe.

**Diagnosis** — The bright yellow or saffron colour is different from the yellow colour generally seen in jaundice. Moreover the sclerotics remain white and the urine and stools are of normal colour. The uramæsis and spectral analysis of urine for picric acid etc. exclude the ordinary toxic pigmentations. The condition is distinguished from certain types of *chromidrosis* as the sweat is not coloured and the clothes do not become stained.

legs and are permanent.

**Treatment** — The only successful measure seems to be to send the patients up country. On the hypothesis that the condition might possibly be of parasitic origin a formalin spirit lotion (1 per cent) was prescribed in several cases, but the improvement if any was very slight, though certain patients stated that they thought the condition was affected in a beneficial manner by it.

### Melanonychia

**Synonyms** — Black pigmentation of nails. *Melanonychia factitia*.

Castellani has described in two European ladies a peculiar condition of the nails characterized by a band of black pigmentation along the free border of the nail. On superficial examination it has the same appearance as though some dirt had accumulated beneath the free border of the nail, but on scraping this pigment that the condition is due to the nail. The sufferer has any internal or external



PLATE XV.



XANTHODERMA AREATUM

## • DYSTROPHILS

medicine which could account for the pigmentation and the  
apart from this line of pigmentation appeared perfectly normal  
We have later come across a case in a European gentleman  
another case in Macedonia  
The condition slowly disappears spontaneously

## Xanthoderma Areatum

This affection which has been described by Castellan is  
infrequently met with among Europeans it generally affects the  
lower parts of the legs it starts very insidiously with a yellowish  
or reddish yellow spot which is not elevated the surface is smooth  
not furfuraceous there is no infiltration and apart from the  
colour the affected skin is normal There is no pruritus and  
no pain The yellow spot slowly increases and one or more other  
spots may appear near the first one or at a distance Some of the  
irregular or various outline forming a large yellow red patch of  
the colour are normal being of normal consistency and elasticity  
The disc is very chronic The general health is not impaired  
the lymphatic glands are not enlarged and the blood does not show  
any abnormality urin normal In all our cases syphilis could be  
excluded in none was there any history of traumatism

**Diagnosis**—From chloasma Xanthoderma areatum is readily  
differentiated by the lighter yellow or yellowish red and by the  
different situation The affection can be easily distinguished from  
Xanthoma as the texture of the skin is normal and the patches  
are not elevated In papular Xanthoma the patches of buff coloured infiltration  
an eruption consisting of small patches of buff coloured infiltration  
are lumpy in some places in others linear It must be distinguished  
also from Schamberg's so-called cayenne pepper condition  
characterized by the presence of brownish yellowish patches  
on the legs made up of small puncta giving rise to a cayenne-like  
appearance of the skin found at times on people suffering from  
varicose veins

**Treatment**—This is difficult in some cases an energetic exfoli-  
ating treatment by *r. sor.* in past *e. resorcin 3ii ac salic gr xx*  
*Lassar's paste 3ii* improves the condition after the inflammation  
induced by the paste subsides

## Mongolian Spots

*Maculæ neonatales*  
*Maculæ natalium*

**Synonym** Mongolian Spots  
**Definition** Maculæ neonatales characterized by the presence  
of dark bluish spots on the lower facial region not disappearing on  
pressure  
**History**—The first complete description has been given by Baclz,  
who found them almost constantly in Chinese Koreans Japanese  
Malays Later Adachi Ashmead Martinotti Consiglio, and  
others have further investigated the subject Castor and Pink



plantar fold which deepens and extends until it encircles the toe which is finally severed from the foot

**History**—The disease was first described by Da Silva Lima in 1852 Clarke in 1860 in his description of the Gold Coast drew attention to a dry gangrene of the little toe found in negroes in that region and considered it to be a manifestation of yaws. In 1867 Da Silva Lima further studied the disease and with Wucherer described fifty cases of it in Brazil and introduced the term *anhum* in the same year Collas wrote an account of the disease Da Silva Lima ventured no explanation of the disease In 1873 Crombie described the disease as occurring in India in 1876 Pirovano found it in Buenos Ayres and in 1877 Corre mentions it in Réunion

It is reported in the West Indies by Potoppidian in 1879 in North Carolina by Hornaday and Pilman in 188 in Nova Be by Deblenne in 1883 in Western Virginia by Duhring in 1884 This last of a or long th Vle et d ed the disease m cronically

the vasomotor nerves leading to a spasm of the vessels endarteritis obliterans fibrosis of the cutis and rarefying osteitis whereby the  
 of this date there are a number  
 ally mentioned those by  
 ) Mur in 1903 Ashley  
 in 1910

**Climatology**—The disease is known in South America especially in Brazil and the Argentina and also in British Guiana in North America especially in the Southern United States but also though rarely in the Northern and in Canada It also occurs in the West Indies In Africa it is especially well known on the West Coast and particularly in the Gold Coast but it also occurs in

Transvaal  
 also occurs  
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 aring rings  
 at once as

there is nothing to support it

According to some authorities—Le Dantec Da Silva Lima etc—the hereditary factor has a certain importance Da Silva Lima quotes the example of a negro family all the members of which presented the condition

The racial factor has also been given much prominence for the  
 col mong natives and mulattoes

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skin of the little toe is more likely to occur in negroes who often are flat footed. It is more common in males than in females, in adults than in children, and though it can apparently be found at any age, is most common between thirty to thirty five years.

We are inclined to believe that the condition is of parasitic origin, the infection taking place probably through the small superficial lesions or wounds which may be found in people going barefooted.

**Pathology.**—The constant irritation causes the epithelium to proliferate internally and depress the skin and cause the fibrous tissue of the cutis to proliferate. There is also an endarteritis, by which t

the toe,  
bone or  
separate  
bone of  
furrow.

wards into the cutis in which the connective tissue is increased in quantity. The vessels show endarteritis and periarteritis, the sweat glands show proliferation and fatty degeneration of the cells. The bone is in a condition of rarefying osteitis.

Distally to the furrow the joints are effaced, the tissues show fatty degeneration and cedematous infiltration. No organisms can be found.

**Symptomatology.**—The disease is purely local and is not, in our experience, attended by any general symptoms. It begins, as a

small globule surrounded posteriorly by a deep groove, by which it

the fingers

**Diagnosis.**—The diagnosis affords no difficulty, the presence of the constricting furrow being typical. It is easily differentiated from leprotic lesions of the toes by the sensibility being normal

is not progressive

**Prognosis.**—There is no danger to life in the disease

**Treatment.**—The disease is best treated by making a longitudinal cut into the groove, when its progress may be stopped

**Prophylaxis.**—The essentials of the prophylaxis are cleanliness and the wearing of stockings and boots to protect the foot from injury

### Symmetrical Palmar Erythema.

Chalmers in 1899 drew attention to a symmetrical non pruriginous palmar erythema found in B. ... and flec

### Acrodermatitis Vesiculosa Tropica.

**Historical and Geographical**—This skin disease was described by

cases the search for Hansen's bacillus being negative and anæsthesia and other signs of leprosy being absent. No history of traumatism was elicited in our patients

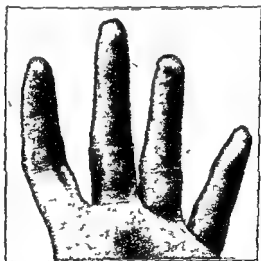


FIG. 879.—ACRODERMATITIS VESICULOSA TROPICA

**Symptomatology.**—In a well marked case the skin of both hands especially the fingers appears glossy and tense the fingers assuming the size of a millet seed the skin of the fingers. criological examination

reveals absence of any bacterium. They may apparently remain unchanged for a long time, then may slowly disappear or a few may

**Course and Prognosis**—The course of the disease extends to several months and occasionally to two or three years with periods of great improvement. Ultimately the condition may get cured spontaneously. The general health is not affected but the patient is unable to work with his hands.

**Diagnosis**—This is based on the patient complaining of severe pains in the hands and fingers, on the presence of deep seated cheiropompholyx like vesicles on the glossy skin and on the long course of the complaint.

The condition is differentiated from cheiropompholyx by the severe pains and absence of hyperidrosis, from a leprotic condition by the absence of

leprosy on other

is long the disease  
dermatitis repens or vitiligo and alopecia areata perstans of Hallopeau by there not being history of traumatism, by absence of exfoliative lesions, by the less severe objective signs and by the absence of the large foci of disease with marked borders and fringed with sodden epidermis which is thrown up by the undermining exudate.

**Treatment**—The regular application of an ichthyol ointment (2 to 5 per cent) to the hands and fingers and the administration of the same drug (gr. iii) three times daily by the mouth is beneficial.

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**Chloasma Symmetricum—Chloasma Bronzium—Xanthoderma Areatum—  
 Dermatosi Foveolata Frontalis**



## CHAPTER LCVIII

### MISCELLANEOUS DISEASES

**Craw-craw**—*Dermatitis nodosa rubra*—*Lichen convex*—Symmetrical ear nodules—Ear lipomata—Porter's lipomata—Subcutaneous nodular lipomatosis—Multiple pruriginous tumours of the skin—*Angiofibroma contagiosum tropicum*—Multiple pruriginous tumours—West Indian nodules—Mossy foot—*Botryomyces*—The hyperkeratoses—Juxta-articular nodules—*Murmelkiasmosis*—References

#### CRAW-CRAW

**Synonym**—Nodular dermatitis (A. Plehn)

**Ætiology**—The cause of the malady is unknown. Pijper has described a diphtheroid bacillus.

**Symptomatology**.—Under the term *craw-craw* African natives denote practically any pruriginous skin disease. Our African experience has taught us that most of the so-called *craw-craw* cases are cases of neglected scabies or of *tinea corporis* or what Daniels and ourselves call *cooly itch*. We apply the term *craw-craw* to a dermatosis met with in Africa in Ceylon and in various parts of

be roundish and flattened and others acuminate when disappeared  
The most of on

taneously

**Diagnosis**—The disease with which *craw* presents the



FIG. 880.—CRAW CRAW

a marked improvement and in many instances a cure. Internal treatment (arsenic ichthyol etc) does not seem to influence the disease.

#### Dermatitis Pruriginosa Tropica (Cooly Itch)

The term *coolyitch* is often applied to dermatoses of various nature including scabies. We use it to denote an extremely pruriginous dermatitis affecting coolies and occasionally Europeans in certain parts of the tropics especially in the low country. No acari or similar parasites are found.

analogy to *Copraitch* (p. 215)

**Symptomatology**—The eruption is generally found on the arms and legs but may extend all over the body even though rarely to the face. The patient complains of unbearable pruritus.

examination may be considered. cunicula are found and no acarus is observed.

**Prognosis**—The eruption is very obstinate and may last for months.

**Diagnosis**—The absence of cunicula and of the sarcoptes differentiates it from scabies.

**Treatment**—Sulphur (3 to 10 per cent) and naphthol ointments (3 to 10 per cent) are very useful though their action as remarked by Daniels is much slower than in scabies.







**DERMATITIS NODOSA RUBRA.**

IN REALITY THE PAPALES ARE OF A BRIGHTER ANGRY RED COLOUR

*To face page 20*

## DERMATITIS NODOSA RUBRA.

**Historical and Geographical**—This condition has been described by Castellani in Ceylon

**Ætiology**—This is unknown

**Symptomatology**.—The first impression received on seeing a patient suffering from this peculiar disease is that he is suffering from smallpox in the papular stage of the eruption but the absence of fever and the closer inspection of the eruption will exclude smallpox at once. In a well marked case the patient presents on his face arms chest back and practically on the whole body numerous

large papules and nodules

The colour of the eruptive

elements is an angry red,

the shape hemispherical or

roundish the size from a

small split pea to a large

pea. The surface of the

papules and nodules is

smooth does not show

umbilication nor scales

their consistency is hard

most of the papules are not

follicular. There is unbear-

able pruritus but the

malady has no urticarial

element whatever. Several

of the superficial lymphatic

glands are enlarged and

hard. In several cases a

well marked enlargement of

the parotid gland is present.

The blood shows a certain

degree of eosinophilia. The

course of the disease is long

—six months to a year and

sometimes much longer. The

nodules become gradually

smaller they leave no scar or

zones of hyper-



FIG 881—DERMATITIS NODOSA RUBRA

nodules become gradually smaller they leave no scar or zones of hyper-

From *acutis* by the unbearable pruritus and by the eruption not being chiefly limited to the face, as well as by the absence of subsequent necrosis

From *follicis* by the condition not being limited to the extremities, and by the absence of central crusts



FIG. 882.—DERMATITIS NODOSA RUBRA

From *erythema multiforme* by the eruptive elements being well

pruritus and the course of the disease is a long one. Occasionally, death ensues

uch affected  
l the severe  
Occasionally,

**Treatment**—Arsenic potassium iodide mercury do not influence the disease To allay the pruritus salicylic alcoholic lotions (2 per cent) and ointments may be used

### LICHEN CONVEX

**Synonym**—Lichen Pilaris Convex (Castellani)

**Historical and Geographical**—This affection—which has been described by Castellani—is very common in Ceylon especially among natives

**Symptomatology**—The regions of the body mostly affected are the thorax dorsum and shoulders The disease is characterized by the presence of numerous firm papules all of which are follicular

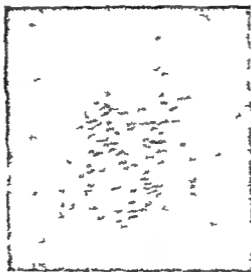


FIG 883.—LICHEN CONVEX

The surface of the papules is smooth and square or plugs are found they have always a convex surface and may be almost hemispheric  $\frac{1}{2}$  to  $\frac{1}{4}$  inch in diameter The colour of the papules has a pinkish hue in natives and red in Europeans they have no inflammatory base they are not surrounded by any halo of inflammation nor is there hyperpigmentation nor do they leave pigmented areas on healing The eruption is very pruriginous.

eral health  
in a few

From *acnitis* by the unbearable pruritus and by the eruption not being chiefly limited to the face, as well as by the absence of subsequent necrosis.

From *folliculitis* by the condition not being limited to the extremities, and by the absence of central crusts.



FIG 882.—DERMATITIS NODOSA RUBRA.

From *erythema multiforme* by the eruptive elements being well-

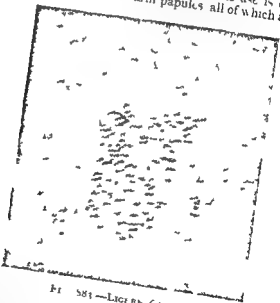
pruritus, and the course of the disease is a long one. Occasionally, after many recurrences death ensues.

## LICHEN CONVEX

**Treatment**—Arsenic potassium iodide mercury do not  
the disease To allay the pruritus salicylic alcoholic lo  
cent ) and ointments may be used

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PL 581—LICHEN CONVEX

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hemispheric shape and are not surrounded by any halo of in  
has a pinkish hue in natives and in Europe they have no  
inflammatory base. The eruption is very pruriginous  
inflammation nor is there hyperpigmentation. The general health  
pigmented areas on healing. The lymphatic glands are not enlarged in a few  
The regions affected may show hyperhidrosis. The general health  
is not affected. The course is long. The eruption  
cases the blood may show a slight degree of eosinophilia.

months and often recurring. When the eruption heals, no hyperpigmentation is left.

Diagnosis—From  *pityriasis rubra pilaris*  by the papules never

skin not presenting a diffuse inflammation whatever the stage of the disease. Moreover, even when the eruption is of long standing, the appearance of the skin between the papular elements remains quite normal and there is no sign of what the French call  *lichenification* .

Prognosis—The eruption lasts for several months, but generally heals spontaneously; recurrences are observed. The general health is not affected.

Treatment—Potassium iodide, mercury, and arsenic have no effect. Externally, antipruriginous lotions and ointments may be used—as for instance, a salicylic alcoholic lotion (2 per cent) followed by a naphthol ointment (2 to 5 per cent). Change to a cool climate is very beneficial. One of our cases improved on a vegetarian diet.

### SYMMETRICAL EAR NODULES

This condition has been described by one of us in Ceylon in 1910, but further experience will probably show that it is to be found also in other tropical countries. In the deep substance of the lobule of both ears—generally the condition is symmetrical—on palpation a spherical nodule, hardly visible, is felt. Now and then the nodule becomes much larger, very tense, and may then present a  
 as a rule no  
 a feeling of  
 of size of the

nodules

The condition is not leprotic, there being no anaesthesia or other sign of leprosy; it may possibly be of parasitic origin, but nothing definite can be stated, as none of our patients would allow the removal of the nodule. Further investigation may show that it is allied to the peculiar condition called Nepal tumour (see Chapter XC, p. 2010).

### EAR LIPOMATA

Symmetrical lipomata of the lobules are not rare (see Chapter XC, p. 2010).

## PORTER'S LIPOMATA

Porters and hammock carriers often show one large lipomatous mass on one or both shoulders where they carry weights or where the pole on which the weight is carried presses

## SUBCUTANEOUS NODULAR LIPOMATOSIS

**Synonym**—Polymicrolipomatosis

This condition seems to be common in the tropics in Europeans and natives alike. It is characterized by the presence of subcutaneous nodules found only on palpation roundish or oval painless the size of a pea to a nut. These are generally situated in the subcutaneous tissue of the arms legs and abdomen and in our

of fatty tissue. Usually the tumours become much larger and may be plainly visible.

## ANGIOFIBROMA CONTAGIOSUM TROPICUM

This disease was first described by Unna and von Bassowitz. So far cases have been reported from the southern regions of Brazil only.

**Symptomatology**—The incubation period varies from fifteen to twenty five days. There are no prodromal symptoms. The eruption consists of vivid red papules which soon enlarge into nodules the size of a large pea to an almond. The eruption may affect any part of the body but more frequently the face neck axillæ and genital organs. It is rarely found on the legs. It very frequently affects the various mucosæ—oral nasal rectal and urethral. The nodules present a smooth surface of a violaceous colour and they are somewhat of soft consistency they bleed severely after the slightest traumatism. They may disappear spon

during sexual intercourse or by the habit the people of Brazil have of taking their maté (national beverage) using the same cannule.

**Histopathology**—According to Unna's investigation the histopathology is quite different from what is observed in frambœsia and syphilis. The nodules consist of fibrous tissue with scanty cells intersected by extremely numerous bloodvessels.

**Diagnosis**—The disease must be distinguished from verruga



with *verruca sensu stricto* (p 1576), by Strong Tyzzer Brues Sellards and Gastiaboru In frambœsia the nodules have a more form surface and do not bleed so easily

Treatment.—Mercury and potassium iodide are useless Basso witz recommends iron and arsenic internally and externally the injection into the base of the nodules of a few drops of formalin, or their excision using the galvano-cautery

### MULTIPLE PRURIGINOUS TUMOURS OF THE SKIN

Schamberg and Hirschler reported in 1905 two cases of multiple tumours of the skin in negroes associated with itching The tumours were sharply circumscribed nodules from the size of a small pea to a large hazel nut situated on the extremities They were of a blackish colour—the smaller smooth the larger covered with a horny epidermis These tumours showed on histological

numerous  
The same  
in a white

woman

### WEST INDIAN NODULES.

This affection which seems to be very similar to the preceding one has been described by Numa Rat in natives of the West Indies, who often confuse it with Guinea worm The eruption may attack any part of the body and consists of subcutaneous nodules varying from the size of a pea to that of a small nut There is extremely severe pruritus and the natives destroy the skin covering the nodules with caustics and extract the nodules which appear yellowish

Histologically accorc  
hypertrophied connective  
bundles The aetiology is unknown

### MOSSY FOOT.

Synonym —Piemugoso

This affection is fairly common according to Thomas in the region of the Amazon

The foot is covered with dense warty very vascular, painful

The suggestion has been made by Cranston Low that it may be a type of tuberculosis cutis verrucosa and by Da Matta that it may be a form of leishmaniasis

### BOTRYOMYCOSIS

Uromyces Granuloma Pyogenicum  
condition characterized by the  
size and collected in clusters.

**Historical and Geographical Distribution.**—The condition was first studied in horses, in which it occurs often in the testicular cord after castration, but it is common also in the pig, dog and cattle. Later it was found in man by Dor and Poncet in Europe

reproduce by a process of endosporulation. The cultures obtained by some authors have, however, all the characters of a staphylococcus

Some authorities, in fact, consider the disease to be merely a type of pyosis due to the usual staphylococci. Magrou, using a special disease from an that th neutral

to be due to an amoeba and his results have been confirmed by Bureau and Labbé but not by others

**Pathology.**—The condition, as seen by us in the tropics has all the characters of a granuloma. The microscopical examination of sections shows young connective tissue with large numbers of plasma cells. Peculiar claviform bodies have been described by Magrou, who believes them to originate from the staphylococci. The lesions are very vascular. Later, denser fibrous tissue is observed

**Symptomatology.**—The condition generally develops on some suppurating wound, but may occur upon the site of any abrasion or wound. It appears as a small, generally cherry-red, granulatous nodule or mass often roundish, of various size—from a pea to a nut or larger. Occasionally the nodule may be pediculated. At first it is of rather soft elastic consistency, later may become fibrous and much harder. There is very little tendency to spontaneous cure. A very mild type of botryomycosis occasionally develops after vaccination. We have seen several such cases

**Prognosis.**—The general health is not much affected, but the condition h

**Treatment.**  
carbolic or pedunculate. It may recur after operation, but this is rare.

**Prophylaxis.**—Care should be taken to keep suppurating wounds thoroughly disinfected.

## THE HYPERKERATOSES

**Definition**—A hyperkeratosis is any cutaneous condition in which the cells of the horny layer have a greater coherence than normal as tested with pepsin and hydrochloric acid and thus tend to pile themselves up in the form of horny scales

D. M. M. A. J. E. J. L. L. — 1

little or no influence on the hyperkeratosis

Further syphilis can certainly act upon the foetus *in utero* and lay the bases of changes of metabolism which may result in the so called congenital hyperkeratosis and the same remarks to a certain extent appear to apply also to tuberculosis

**Classification**—With the above provisos the hyperkeratoses may be classified into —

A *Hyperkeratoses obviously associated with a causal disease* —

1 *Non follicular* —

Found in leprosy arsenical poisoning and hyperidrosis.

2 *Follicular* —

Found in various tubercular and syphilitic affections and some forms of lichen and acne

B *Hyperkeratoses of unknown origin or remotely associated with syphilis yaws tuberculosis etc* —

(a) *Develops during intra uterine life* —

*Hyperkeratosis universalis congenita*

(b) *Develops during post uterine life* —

1 *Generalized affections* —

Of these *ichthyosis* and *psoriasis rubra pilaris* are met with in the tropics

2 *Localized affections* —

(1) *Non follicular* —

(1) Without acanthosis or markedly dilated papillary vessels—*Keratoderma*

(2) Without acanthosis but with markedly dilated papillary vessels—*Angiokeratoderma*

(3) With acanthosis but without markedly dilated papillary vessels—*Acanthokeratoderma*

(1) *Follicular—Keratosis*

In the tropics we have met with leprotic and syphilitic hyperkeratoses as well as with those connected with lichen planus. We have also seen ichthyosis, pityriasis rubra pilaris and keratosis palmaris et plantaris and several other forms, but of all these, three forms must receive a little further notice—viz —

- 1 Keratoderma cribrata
- 2 Acanthokeratoderma præcornuifaciens
- 3 Keratoma plantare sulcatum

### KERATODERMIA CRIBRATA

keratosis of the  
which the hyper  
the shedding of

little corn like projections

**Remarks**—In the tropics it was first described by Castellani and then by Chalmers

**Ætiology**—It seems that it is in some way associated with yaws or syphilis (congenital or acquired) probably by changes effected in the metabolism and not by the action of their parasites

**Pathological Histology**—The essential points are a mild chronic inflammation of the dermis and a hyperkeratosis of the sweat orifices leading to the formation of corn like projections which are freed laterally and finally all round and then fall out leaving a depression

**Symptomatology**—The palms of the hands or soles of the feet may show hyperkeratosis associated with slight itching. In the hyperkeratotic area there are many corn like bodies some of which have fallen out and left depressions. The condition is very chronic

**Diagnosis**—The bilaterally symmetrical hyperkeratosis of the palms or soles with the pits in the thickened areas and the corn like bodies are characteristic

**Treatment**—Nothing is known to permanently benefit the condition

### ACANTHOKERATODERMIA PRÆCORNUFACIENS

This is an acanthokeratoderma characterized by the formation of thickened patches of epidermis in the palms of the hands and soles of the feet which may (in the latter situation) become cracked and

as which prevent the

is associated with a

thus giving rise to a

hyperkeratosis subun

ith syphilis but the

specific organisms cannot be found in the lesions and antisyphilitic

may last for months and if the patient has much walking to do may become very painful

improvement

### JUXTA-ARTICULAR NODULES.

Synonyms — Enno <sup>3</sup> Naridé <sup>3</sup> Macgregor's nodules Steiner's <sup>3</sup> affection was first briefly

a possible  
Steiner in  
it is now  
term of

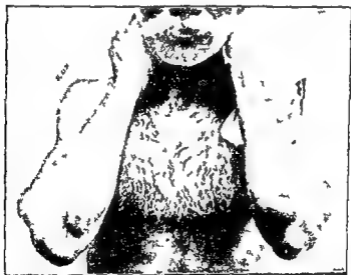


FIG 887—JUXTA ARTICULAR NODULES

Madagascar considered the cause of the affection to be a fungus *Nocardia carougeau* Brumpt 1910 Cases have been observed in Chalmers and y be found to es

**Ætiology** — Macgregor Steiner and juxta articular considered the nodules to be of parasitic origin while Fontoyneont and Carougeau found a fungus which they believed to be the ætiological agent of the condition The description of this fungus—*Nocardia carougeau*

Brumpt 1910—is given in the chapters on fungi (see pp 1065 and 1066) Recently the presence in the nodules and etiological rôle

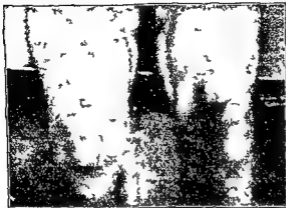


FIG 888—JUXTA ARTICULAR NODULES

of this fungus has been doubted by many authorities. Several observers consider the condition to be a late manifestation of framboesia.

**Symptomatology**—In the legs and arms especially in proximity to the articulations several nodules are found some of them the size of a walnut or more of rather hard consistency and covered by healthy skin. According to Macgregor these tumours occur most frequently about the elbows or the parts of the body coming in contact with the ground when the native is sleeping. These nodules at first are rather soft and are situated in the subcutaneous tissue and the skin may be moved above them. Later they may apparently fuse together forming hard large tumours and adhere to the skin which generally does not present any alteration. The course is very chronic.



FIG 889—Juxta articular nodule

**MURMEKIASMOSIS AMPHILAPHES.**

Chalmers and Christopherson have described a case (Fig' 88c) of spreading warts associated with *Cryptococcus myrmeciae* which grew on the skin of the face and neck destroyed an eye and entered the mouth

**LYMPHO-FIBROMATOSIS**

This is a condition of elephantoid fibrosis (Fig 890) met by us in Ceylon and Africa It is sometimes associated with a secondary



FIG 890 —LYMPHO-FIBROMATOSIS

pyogenic eruption : Some cases may be associated with filariasis but others are not The skin is elevated into large raised flattened patches of fibrous consistency The condition is chronic

**SEBORRHŒA SPINULOSA.**

This condition has been seen by Castellani and Chalmers in various parts of the tropics and the Balkans It is characterized by the presence of numerous yellow plugs some of which are acuminate and hard These plugs project from the orifices of the sebaceous ducts and are often situate on an oily skin Ordinary black comedones are absent The term 'seborrhœa spinulosa' probably

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## CHAPTER XCIX

### COSMOPOLITAN SKIN DISEASES

General remarks—Pyogenic infections—The erythemata—The exanthemata

#### GENERAL REMARKS.

found in the high mountainous regions of the tropics

The diagnosis of some of the cosmopolitan dermatoses may, owing to the colour of the skin of native races be very difficult to the

at coloured races are on the whole less liable to ... than the white races, but the reverse is more in accordance with our experience

A few remarks as to the normal skin of native races may be useful before proceeding to review the various cosmopolitan diseases. In the African races and American negroes who descend from African negro stock (*Guinea negroes Yolloffs Caffres*) the whole skin especially the derma is thicker than in the white races. In Indian races the skin is about the same thickness as in Europeans except in the Tamils whose skin is somewhat thicker. As noted by Howard Fox, the glandular system—sweat glands and sebaceous glands—is much more highly developed in the native races especially the African negroes. The sebaceous secretion is the cause of their peculiar odour and the shining appearance of the skin and the large secretion of sweat, which owing to the high temperature, quickly evaporates is the probable cause of the skin feeling cooler when touched. In native races the hairy system is less developed except on the scalp, in the African races the hair of the head is generally curly or woolly, in Indian races it is usually smooth

mentation is found on the prepuce, vulva, palms, and soles. In  
 mentation in Erythrodermia, we have not noted any distinct difference,

ally the  
 e white  
 in the  
 ordinary dolorific sensibility, but the thermic sensibility is probably  
 less

### PYOGENIC INFECTIONS.

These are very common in tropical countries. Impetigo and  
 Ecthyma lesions are frequently met with. The symptoms and  
 course are identical with what one finds in temperate zones, and  
 the treatment is the same—removal of the crusts, disinfection with  
 a lotion, such as a perchloride of mercury (1 in 2,000), and dressing

from the patient. When the crusts are removed, the lesions

become absorbed.

As a preventive a salicylic alcoholic lotion (1 to 3 per cent) used after the daily bath is advantageous

**Sycosis coccigenica**—This is fairly common in both Europeans and natives. In the negroes Fox has often observed the formation of tiny cheloidal tumours after this affection. Depilation and the use of a vaccine is the best method of treatment. We have observed a case of *Dermatitis papillaris capillitis* (Kaposi)

**Erysipelas**—Ordinary erysipelas due to streptococci and to be distinguished from the filarid erysipelatoid attacks preceding the development of elephantiasis is not rare. Ichthyol ointment or lotion (20 per cent) answers well

## THE ERYTHEMATA

**Erythema solare** is common in Europeans recently arrived and in those who live an open air life such as planters. It is followed by pigmentation (sunburn see pp 82 and 2231). The application of calamine lotion followed by the use of boric vaseline or rose-ointment is beneficial



FIG 891—HERPES IRIS

**Erythema intertrigo** is very frequently observed in corpulent persons. Washing the parts with a potassium permanganate solution (1 in 5000) followed by application of boric talc or salicylic talc powder (acid boric ʒi talci ʒi or ac salicyl gr x talci ʒi) is useful. Persons suffering from intertrigo are very liable to become infected with *trichomycosis*

**Erythema nodosum**—**Erythema multiforme**—These affections are occasionally met with but much more rarely than in temperate zones. We have seen two cases of the variety of erythema multiforme known as *herpes iris*

from fever and coming from the suspicion of trypanosomiasis

**Diffuse Erythema scarlatiniforme** and **Erythema morbilliforme** may be seen in some malarial patients. They may occasionally be

**Purpura**—Schonlein's purpura Henoch's purpura and Werlhof's purpura are not common in the tropics though we have seen cases. A symptomatic purpura eruption is occasionally seen in the last stage of kala azar in malaria and other maladies.

### THE EXANTHEMATA.

Smallpox chicken pox and measles are very common in the tropics though scarlet fever is but rarely met with.

**Smallpox**, of which we have already given a description (p 1486) is very common and often spreads in extensive epidemics and may present the confluent type and the hæmorrhagic type which is rare in Europe. In colonies under European rule vaccination is extensively practised. The vaccine does not retain its immunizing properties for more than a few weeks in the tropics and hence it is advisable for each colony to be provided with a central vaccine depot where the lymph can be prepared under careful supervision. Owing to the habits of the lower classes and to many of the vaccinators being non medical men serious infections with other diseases may occur.

**Measles** may be difficult to recognize as the eruption in dark skinned races is better felt than seen but the coryza and other symptoms are generally sufficient to enable a correct diagnosis to be made.

### URTICARIA—LICHEN URTICATUS—PRURIGO

*Hæmorrhagic urticaria*—

201  
sl:  
se  
ur:

We have observed true prurigo of Hebra in two native girls.

### DERMATITIS VENENATA

Several tropical plants and grasses produce substances highly irritating to the skin but the subject has already been treated in

animal agents such as ants bugs caterpillars etc have been already mentioned in Chapters XIV and XCVI p 2200

### PARASITIC DISEASES

**Trichophytoses**—In tropical countries in addition to the trichophytoses peculiar to the tropical climate several other forms of trichophytic affections occur which are clinically identical with those met with in temperate zones such as *tinea circinata* *tinea*

capitis tinea barbæ The fungi are, however in most cases different species (see p 2052) In our experience, tinea capitis is less common in India Ceylon and tropical Africa than in Europe and America On the other hand it is extremely common in the American negroes The fungi found in the tropics are generally large spored ones *Microsporium audouini* has never

been found by us *Favus* is rare in tropical Africa and Asia but common in China Egypt and in the Sudan

#### Pityriasis versicolor—

The pityriasis versicolor of temperate zones may be found in the tropics It is to be noted, however, that several writers on tropical medicine confuse pityriasis versicolor with another dermatomycosis which is extremely common—tinea flava (see p 2073)

#### Pediculosis and Scabies

—*Pediculosis capitis* is extremely common although natives generally take great care to keep their hair in good condition by regularly combing, washing and oiling it According to Fox Carmichael and Pendergast pediculosis capitis in the Southern States of North America is much less frequent in the negroes than in the whites, as according to them the negroes take greater pains in the care of the scalp than do the



FIG 892—SEPTIC PEMPHIGUS

lower class whites *Pediculosis corporis* and *Pediculosis pubis* are as frequent in the tropics as in temperate zones and so is scabies *Pediculosis pubis* may however be very rare or absent in races accustomed to regular shaving of the pubic hair For description of the parasites see p 753 Scabies is described on p 2217

## BULLOUS ERUPTIONS.

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## HERPES.

us is characterized by a sharp attack of itching and burning



FIG 893.—PSORIASIS

and natives.

**ECZEMA—ACRODERMATITIS PERSTANS—PSORIASIS—  
PARAPSORIASIS—SEBORRHOEA—DERMATITIS EXFOLIATIVA**

Eczema is extremely common and all varieties of it are found in the tropics as in temperate zones both in natives and Europeans. The treatment is often very difficult. In our experience greasy preparations and even pastes are as a rule badly borne except in some chronic cases. Generally speaking it is best to use lotions

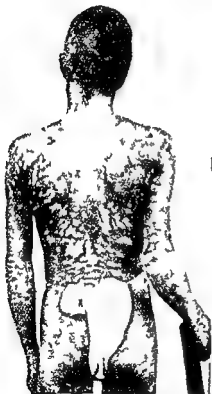


FIG 894.—PSORIASIS IN A SINHALESE

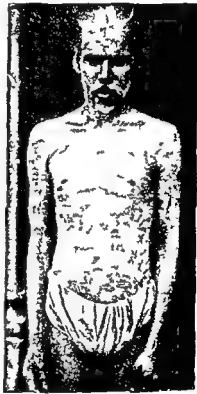


FIG 895.—DERMATITIS EXFOLIATIVA  
IN A SINHALESE

such as resorcin ( $\frac{1}{2}$  to 1 per cent) or liq. plumbi (2 per cent). Some cases of eczema will improve only on the patient going to a cooler climate. Wilson's dryness and hardness is frequently met the toes.

**Acrodermatitis Perstans**—We have seen two cases of a very obstinate chronic dermatitis with exfoliation on the extremities

of Sinhalese natives apparently identical with acrodermatitis perstans of Hallopeau. They were different clinically from the acrodermatitis vesiculosa described by one of us.

Psoriasis is considered by most writers to be extremely rare or absent in native races in the tropics. Rutz states that he never

observed it during his many visits of  
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liarity. In our experience psoriasis is frequently met with in the tropics in Europeans and natives of every race.

Seborrhœic affections are common in the tropics especially in Europeans. A seborrhœic rash of the chest is frequently met with and often confused with ring worm. The microscopical examination will clear the diagnosis. The use of a sulphur ointment (5 per cent) is the best treatment.

In Ceylon a peculiar form of seborrhœa capitis with yellowish scales is occasionally seen in European children between four and twelve years of age. This affection is probably due to *Pityrosporum canis* Castellani, 1908 and quickly disappears when the child goes to cooler climates.

Dermatitis exfoliativa (pityriasis rubra) is not rarely met with occasionally following on psoriasis. In Ceylon we have observed two cases in Sinhalese natives. The amount of desquamation was enormous. On removing the scales which were white the skin appeared of an angry red colour. At times on superficial examination certain cases may be mistaken for diffuse tinea imbricata.



FIG. 896.—DERMATITIS EXFOLIATIVA SIMULATING *TINEA IMBRICATA*



### ACNE VULGARIS—ACNE ROSACEA—ACNE VARIOLIFORMIS— ACNE CHELOID

*Acne vulgaris* and *Comedos* are extremely common in all races. Both types of *acne*—*Acne indurata* and *Acne papulosa et pustulosa*—are observed. The treatment which is as difficult in the tropics as in temperate zones is based on the use of medicated soaps and sulphur or ichthyol lotions or ointments. The vaccine treatment may occasionally give good results.

*Acne rosacea* is in our experience comparatively rare in the tropics though we have seen cases in all races. True *rhytrophima* we have also observed.

*Acne Varioliformis*—This in our experience is as frequent in the tropics as in temperate zones and may be found in all races. The regions of the body more commonly affected are the forehead, temples, the front of the sternum and the interscapular region.

*Acne cheloid* is very common especially among native races. The back of the neck is generally affected. Cases of *Acnitis* and *Folliculitis* are occasionally seen. These are probably tuberculides.

### HYPERIDROSIS AND BROMIDROSIS

These conditions are for climatic reasons very common in the tropics and have already been considered (see p. 2272). The treatment may be very difficult. Naphthol or salicylic alcoholic lotions (1 per cent) followed by a salicylic boric, or tanniform powder are useful. If there is much inflammation of the skin no

undergarments. Internal treatment by sulphur acid drinks etc. is not of much use. Belladonna and atropin will stop the secretion for a time but they must be pushed till unpleasant symptoms occur. We have seen a case of *chromidrosis* of the axilla in a native boy due to the presence of a bacillus closely allied to the *Bacillus prodigiosus* and another due to a red pigment producing coccus.

*Granulosis rubra nasi* is occasionally seen in half caste and European children.

### LICHEN PLANUS—LICHEN SPINULOSUS—PITYRIASIS RUBRA PILARIS—PARAKERATOSIS VARIEGATA—POROKERATOSIS

*Lichen planus* is common in the tropics among Europeans and natives of the various races. In very dark skinned natives the diagnosis may be difficult to the medical man used to seeing skin

diseases in Europeans only. Apart from the colour however the skin lesions are identical the papules having an angular outline

**Parakeratosis variegata** was seen by us in a half caste patient. We have come across two cases among natives of Mibelli and Resphighi. **Porokeratosis**. We have seen a case of **Granuloma annulare** in a European planter and one in a native clerk.

### TUMOURS OF THE SKIN

The tumours met with in temperate zones are met with also in the tropics in Europeans as well as natives. There is no doubt however that native races are more subject to some classes of tumours than to others. We would call attention to the extreme frequency among the natives of cheloid, the common occurrence of fibroma molluscum and the comparative rarity of epithelioma of the face.

### CONNECTIVE-TISSUE TUMOURS

#### Benign Connective-Tissue Tumours

**Simple fibroma**, **Fibroma pendulum**, and **Fibroma molluscum** (neuro fibromatosis of Recklinghausen) are very frequently met with in natives. **Myomata** are occasionally observed—in our cases always on the face.

**Angiomata** are not rare and multiple **Telangiectases** are fairly common and **Lymphangiomata** very frequent.

**Xanthoma planum** and **Xanthoma tuberosum** are frequently observed.

**Xanthoma diabeticorum** is very common in India and Ceylon where the better classes suffer greatly from diabetes. An example of **Balzer's Pseudo Xanthoma** or **Elastorrhaxis** affecting the abdomen was observed by us in a half caste woman.

**Cheloid**—This is extremely frequent in native races. According to some authors negroes suffer from it sixteen to eighteen times as much as whites. In Indian races it is not so frequent as in African natives, but still much more frequent than in



FIG 097—CHELOIDS

Europeans. The smallest wounds such as those made in tattooing may be followed by cheloid. In Chinamen it often follows hypodermic injections of morphia.

#### Malignant Connective-Tissue Tumours.

Multiple sarcomatosis of the skin is rare. We have seen two cases both in Sinhalese girls.

Mycosis fungoides has been observed by us once only in a half-caste man.

### EPITHELIAL TUMOURS.

#### Benign Epithelial Tumours

Epithelial moles are frequently observed.

Molluscum contagiosum is met with in all races.

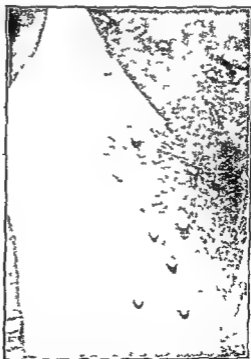


FIG 898 —MOLLUSCUM CONTAGIOSUM

Verrucæ and Warts are extremely common. In two instances we have seen Sinhalese boys covered all over the body with hundreds of warts. Filiform warts are met with and warts on the genital organ.



FIG 899—MOLLUSCUM CONTAGIOSUM HISTOLOGICAL FEATURES

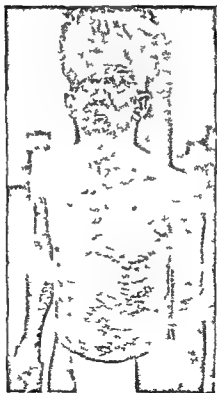


FIG 900—TIPROMA MOLLUSCUM 1 A SITHALLE

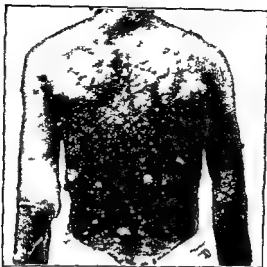


FIG 901—FIBROMA MOLLUSCUM LARGER VARIETY



FIG 902—FIBROMA MOLLUSCUM LARGER VARIETY

are very common. *Verruca senilis* and *Verruca seborrhoeica* are frequent. The best treatment for ordinary warts is carbon dioxide snow.

Escomel has drawn attention to a peculiar treatment for warts in vogue among certain races in South America. These people apply the crushed body

of a beetle belonging to the genus *Meloe* to the wart the horny layer of which has been first scraped off. The wart disappears leaving a slight whitish scar. The active principle is to be found in the beetle's blood.

It is observed also in natives going to *culancum* the same condition (862 p 2187)

### Malignant Epithelial Tumours.

Very rare among natives but we have observed a case of disease of the nipple among

Small Cases

### MIXED TUMOURS

*Adenoma sebaceum* of the face has been seen but rarely by us. We have never observed in the tropics cases of true *Angiokeratoma* on the hands but we have seen a somewhat similar tumour on the scrotum of a European. A diffuse type of *angiokeratoma* of the foot somewhat resembling *mycetoma* has been described in South America (see p 2147)

### RHINOSCLEROMA

A case of *rhinoscleroma* has been observed by us in an Indian coolie and Gros has reported the occurrence of the disease in Algiers.

### TUBERCULOSIS AND TUBERCULIDES—LUPUS ERYTHEMATOSUS

*Lupus vulgaris* is met with in the tropics in all races but according to our observation is far from being so common as in Europe.

*Tuberculosis verrucosa cutis* is very rare and *Scrofuloderma* much less common than in temperate zones. All the so-called tuberculides—*Lichen scrofulosorum*, *Aene scrofulosorum*, *Bazin's Erythema induratum*, *Follicellitis*, and *Aenitis*—are observed but rarely in the tropics.



FIG 903—LUPUS VULGARIS IN A SINHALESE

**Lupus erythematosus**, as regards which we agree with those writers who do not consider it of tubercular origin is very rare in the tropics

While tuberculosis of the lungs is at the present time extremely common in many parts of the tropics skin affections of tubercular origin are comparatively speaking infrequently met with

### SYPHILIS—VENEREAL SORES—BALANO-POSTHITIS

At the present date syphilis is rampant all over the tropics. In the past there is reason to believe that it was unknown in many of the tropical regions which were unopened to the cosmopolitan



FIG. 904.—PAPULO SQUAMOUS SYPHILIDE

trade. According to Lambkin in some parts of Uganda syphilis affects more than half of the population and this enormous diffusion of the malady has taken place during recent years since the country

## SYPHILIS

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FIG 905—POSTULAR SYPHILIS

marked in natives and circinate and pustular syphides, as  
 served by H Fox are more common. The tertiary ulcerat

nitive races the so called parasyph ... ..  
 paralysis and tabes dorsalis) are extremely rare

As regards the treatment of syphilis in the tropics injections  
 very unpleasant in a hot damp climate. We generally prefer



been given in the chapter on Iramboesia p 1560 We use also injections of various preparations of mercury A mixed mercury and potassium iodide treatment is of advantage in some cases Natives are said by some authors to be extremely susceptible to mercury but in our experience this has been exaggerated We



FIG 906—ULCER TERTIARY  
SYPHILIS



FIG 907—ICHTHYOSIS IN A  
SINHALESE GIRL

generally give them the same doses as to Europeans Native doctors give often mercury disguised in various ways Decoctions of various herbs are also administered In Abyssinia and Erythraea a decoction of a herb related to sarsaparilla and called *usciva* is much used according to Annaratone

**Venereal Sores**—These are common in all races but are more apt to take a phagedænic character in the tropics

**Balano-posthitis**—Every type of balano posthitis is met with in the tropics

**ICHTHYOSIS—SCLERODERMIA—MORPHEA.**

Every type of Ichthyosis is met with in the tropics, from simple Xerodermia to Ichthyosis hystrix. Diffuse Sclerodermia is rare



FIG 908—Ichthyosis

but circumscribed sclerodermia (morphea) is rather frequent We have seen a case of Sclerema neonatorum in a Sinhalese baby

**KAPOSI'S DISEASE—ACANTHOSIS NIGRICANS—DARIER'S DISEASE.**

We have seen in the tropics several cases of Kaposi's disease

Sinhalese beggar

**LENTIGO—CHLOASMA.**

Freckling cannot be seen in very dark skinned natives, but is easily visible in half castes It is very common among Europeans especially those who live an open air life and are much exposed to the sun.

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## BIOTRIPSIS.

Cheate has described under the name 'biotripsis' or 'life-wear,' the trophic changes which take place in the skin of old people. In Europeans the skin especially of the hands, may become at places shiny smooth melastic more or less pigmented than normal and scar like lesions may be present. Castellani has described a somewhat similar condition in old Sinhalese in whom the skin on the



FIG 909 — BIOTRIPSIS IN AN OLD SINHALESE MAN SIMULATING A MILD TYPE OF ICHTHYOSIS.

legs may present a peculiar condition in which the superficial layers crackling in flexion might be taken for probably a trophic condition due to old age. Castellani has given an account of this condition as seen in the Sudan.

## DISEASES OF THE HAIR AND NAILS.

The diseases of parasitic origin are common and have been described. Cases of *Hypertrephosis* in man and woman are occa-

hair long and do not take care of it, we have seen a condition of inextricable matting of the hair somewhat resembling *Plica*.

Canities is said to take place at a later age in negroes than in Europeans but in our experience neither in negroes nor in Indian races is there any distinct difference. Alopecia of every origin is met with in the tropics. Alopecia seborrhœica is very frequent among Europeans and educated natives. It is rare among the low classes and coolies. Alopecia senilis is less frequent in natives than in Europeans. Alopecia areata is apparently rare among natives but recently we have come across several cases. It is fairly frequent among Europeans. We have seen a case of pseudo pelade of Brocq (folliculitis decalvans) in a Sinhalese man.

All the diseases of the nails met with in temperate zones are to be found also in the tropics. Brittle nails (onychorrhæxis), ridged nails, transverse furrowing, and thinning of the nails, eggshell nails, shedding of the nails, are conditions occasionally seen. Leuconychia, or whitening of the nails either in spots (leuconychia

so called nail pterygium, or outgrowth of the posterior nail fold is occasionally seen. or the opposite condition exposure of the root of the nail (flous unguium) may be met with.

We have never observed a case of congenital atrophy but we have seen one case of supernumerary nails. We have noted with

and those of the toes

Onychomycosis of various origin is a common affection and has already been considered (p. 2059)

### SOME COSMOPOLITAN DISEASES OF MUCOUS MEMBRANES

D. L. F. T. M. H. C. S.

which by some writers have been described as a sign of ankylostomiasis. These pigmented patches are roundish or oval and

natives and is simply due to chewing betel. The pigmentation slowly disappears on the native discontinuing the use of betel. Cases of furrowed tongue (scrotal tongue) are not rare. We have seen a case of Fordyce's disease (pseudo colloid of the lips) in a half caste. A case of *cheilitis exfoliativa* in a European lady and cases of *perlèche* have been observed by us among European children. Under the term *seasonal recurrent ulceration of the lips* Gros has described a very superficial ulceration on the lower lips in Algerian natives which is very common in the hot season and is due according to him to a diplobacillus.

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